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ENVIRONMENTAL ASSESSMENT BOARD

VOLUME: 122

DATE: Friday, August 11th, 1989

BEFORE: M.I. JEFFERY, Q.C., Chairman

E. MARTEL, Member

A. KOVEN, Member



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HEARING ON THE PROPOSAL BY THE MINISTRY OF NATURAL
RESOURCES FOR A CLASS ENVIRONMENTAL ASSESSMENT FOR
TIMBER MANAGEMENT ON CROWN LANDS IN ONTARIO

IN THE MATTER of the Environmental
Assessment Act, R.S.O. 1980, c.140;

- and -

IN THE MATTER of the Class Environmental
Assessment for Timber Management on Crown
Lands in Ontario;

- and -

IN THE MATTER OF a Notice by the
Honourable Jim Bradley, Minister of the
Environment, requiring the Environmental
Assessment Board to hold a hearing with
respect to a Class Environmental
Assessment (No. NR-AA-30) of an
undertaking by the Ministry of Natural
Resources for the activity of timber
management on Crown Lands in Ontario.

Hearing held at the Ramada Prince Arthur
Hotel, 17 North Cumberland St., Thunder
Bay, Ontario, on Friday, August 11th,
1989, commencing at 8:30 a.m.

VOLUME 122

BEFORE:

MR. MICHAEL I. JEFFERY, Q.C.	Chairman
MR. ELIE MARTEL	Member
MRS. ANNE KOVEN	Member

A P P E A R A N C E S

MR. V. FREIDIN, Q.C.)	MINISTRY OF NATURAL
MS. C. BLASTORAH)	RESOURCES
MS. K. MURPHY)	
MS. Y. HERSCHER)	
MR. B. CAMPBELL)	MINISTRY OF ENVIRONMENT
MS. J. SEABORN)	
MR. R. TUER, Q.C.)	ONTARIO FOREST INDUSTRY
MR. R. COSMAN)	ASSOCIATION and ONTARIO
MS. E. CRONK)	LUMBER MANUFACTURERS'
MR. P.R. CASSIDY)	ASSOCIATION
MR. H. TURKSTRA	ENVIRONMENTAL ASSESSMENT
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MR. R. EDWARDS)	NORTHERN ONTARIO TOURIST
MR. B. McKERCHER)	OUTFITTERS ASSOCIATION

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MS. B. LLOYD)	
MR. J.W. ERICKSON, Q.C.)	RED LAKE-EAR FALLS JOINT
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MR. S.M. MAKUCH)	
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MR. G.J. KINLIN	DEPARTMENT OF JUSTICE
MR. S.J. STEPINAC	MINISTRY OF NORTHERN DEVELOPMENT & MINES
MR. M. COATES	ONTARIO FORESTRY ASSOCIATION
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MR. M.O. EDWARDS	FORT FRANCES CHAMBER OF COMMERCE
MR. P.D. McCUTCHEON	GEORGE NIXON
MR. C. BRUNETTA	NORTHWESTERN ONTARIO TOURISM ASSOCIATION

I N D E X O F P R O C E E D I N G S

Witness:

Page No.

PETER KINGSBURY,

LEONARD RITTER, Resumed

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Continued Cross-Examination by Ms. Cronk

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I N D E X O F E X H I B I T S

<u>Exhibit No.</u>	<u>Description</u>	<u>Page No.</u>
717	Photocopy of abstract re: farmer mortality study provided by Dr. Ritter.	20462
718	Extracts from World Health Organization Report. (Reserved)	20476
719	Extracts from paper by Mr. Kingsbury. (Reserved)	20477
720	News Release from Ontario MOE dated April 16, 1987.	20478
721	Article entitled: Assessment of the Potential of Insecticides, Emulsifiers and Solvent Mixtures to Enhance Viral Infection in Cultured Mammalian Cells published in Applied and Environmental Microbiology.	20482
722	Article entitled: Browse Availability After Conifer Release in Maine's Spruce/Fir Forests by Newton, et al.	20528
723	Article entitled: The Fate of Glyphosate in an Oregon Forest Eco-system by Newton, 1984.	20536
724	Paper entitled: The Determination of Persistence, Movement and Degradation of Hexazinone in Selected Canadian Boreal Forest Soils.	20560

1 ---Upon commencing at 8:30 a.m.

2 THE CHAIRMAN: Thank you. Good morning.

3 MS. CRONK: Mr. Chairman, just before we
4 begin, I have had some brief discussions this morning
5 with Mr. Freidin about scheduling matters and perhaps
6 it would be appropriate to raise it now.

7 I'm wondering if Mr. Freidin and Ms.
8 Murphy are in a position to inform the Board and other
9 counsel as to their best estimate today as to when
10 their case might conclude, taking into account the
11 Dryden satellite location hearing.

12 I have, of course, a number of reasons
13 for raising that, given that our case is to commence
14 immediately after the evidence of Dr. Baskerville which
15 will follow the MNR's case, and perhaps I could tell
16 you what the concerns are from our point of view after
17 we hear about the scheduling projections from Mr.
18 Freidin.

19 THE CHAIRMAN: Okay. Mr. Freidin?

20 MR. FREIDIN: Our best estimate is that
21 we would complete our case on November the 15th. I can
22 give you a brief idea of how I arrive at that.

23 It's primarily another week for this
24 panel, three weeks of cross-examination on Panel 14,
25 which is what the indication was before we broke;

1 starting Panel 15 on the 11th of September, taking a
2 week perhaps in direct; a week of cross-examination;
3 off to Dryden until the end of September; two more
4 weeks of cross-examination on 15 which would take us to
5 October the 15th; and then three weeks for Panels 16
6 and 17, both direct and cross-examination.

7 Giving myself a week leeway on that
8 schedule, it takes us to November the 15th.

9 THE CHAIRMAN: And I guess there is no
10 indication at this time how long Baskerville might be?

11 MR. FREIDIN: Only the comment made by
12 Mr. Turkstra the other day that he thought that the
13 examination could be a week to two in direct, as I
14 recall his comment. Other than that, I have no idea
15 how long it might take.

16 THE CHAIRMAN: And in December we will be
17 rising prior to the 15th of December. When I say
18 prior, not later than, until the first week in January
19 when we could recommence. So it looks unlikely that we
20 would finish Dean Baskerville before the break.

21 MR. FREIDIN: I think if we wanted to
22 ensure that we have set enough time aside for him, I
23 think you are right. But that's the best estimate that
24 I can give, Mr. Chairman, and perhaps that would be
25 helpful to Ms. Cronk and she could make her submissions

1 based on that information.

2 MS. CRONK: It was in part for those
3 reasons that I raised it, Mr. Chairman. Obviously --
4 well, one of the first panels that our client -- will
5 be presented on behalf of our clients is comprised of
6 senior executives from a number of the industry
7 companies.

8 Their schedule, like everyone else's, and
9 the many experts who have appeared before you, are
10 difficult to deal with. They are particularly
11 difficult to deal with when they spend a great deal of
12 time out of the country or in other locations in the
13 country, so we were concerned about being able to
14 assure that they were here when they were required to
15 be.

16 When I knew the November 15th date that
17 was being projected and then heard Mr. Turkstra
18 indicate that the examination in direct of Dean
19 Baskerville might take as long as one or two weeks, it
20 occurred to me, sir, likely, although of course not
21 probable, that our case would not in the normal course
22 be called upon to start until the beginning of the new
23 year.

24 It would be of enormous assistance to us
25 in our preparation and as well to arranging the

1 scheduling of a large number of people, if the Board
2 and other counsel are in a position to do so, to
3 indicate that we could assume that we will not be
4 starting until the first week of January.

5 I recognize that on occasion things have
6 been abbreviated during the course of the evidence here
7 so that things are moved up, but regrettably that's not
8 historically the direction it has gone in.

9 So I would think that it might be an
10 overage as opposed to underage, but you understand the
11 problem, sir.

12 What I'm really saying is, that if we
13 could obtain a fixed date, recognizing the best
14 submissions you have heard, it would help enormously in
15 the scheduling and also our ability to ensure that
16 people whose schedules are most demanding are here when
17 they are needed.

18 ---Off the record discussion

19 MS. CRONK: Ms. Murphy was just pointing
20 out as well, Mr. Chairman, the psychic as she sometimes
21 is, that a fixed date assists in determining the time
22 for delivering evidence packages.

23 THE CHAIRMAN: Well, Ms. Cronk, I think
24 the Board can assist you in this way: Based on the
25 best estimates it looks very much like we won't be able

1 to start with your case until the beginning of the new
2 year realistically.

3 Should, by some fluke, history be
4 rewritten so that we do in fact finish with Dean
5 Baskerville earlier and there is some time that the
6 Board would want to utilize, then perhaps we will give
7 consideration to another satellite hearing.

8 And as long as we have enough notice
9 period for that hearing - and we may be in a better
10 position as we go down the track - we may be able to
11 plug in another one at some stage if we have the
12 available time.

13 Looking at the calendar, January the 1st
14 comes out, I think it is on a Monday.

15 MR. MARTEL: Monday.

16 MR. CASSIDY: That's correct.

17 THE CHAIRMAN: And, consequently, it
18 would be unlikely that we would start until the
19 beginning of the following week. And you have chosen I
20 believe to present your case in Thunder Bay; is that
21 correct?

22 MS. CRONK: That's correct.

23 THE CHAIRMAN: So we would then
24 recommence in the new year up here on the second Monday
25 in January--

1 MS. CRONK: Thank you.

2 THE CHAIRMAN: --with your case.

3 MS. SEABORN: Mr. Chairman, would it be
4 possible to consider starting on the Tuesday of that
5 week? I only suggest that because we will not have
6 been in Thunder Bay for some time and it will take
7 ourselves at least some time to get organized and
8 collect exhibits and set up our office, so to speak,
9 for that portion of the case.

10 ---Discussion off the record

11 THE CHAIRMAN: All right. The Board
12 would consider starting on the Tuesday but sitting that
13 Friday of that first week and that will give everyone
14 an opportunity to get organized the first day back.

15 MS. CRONK: Thank you very much, Mr.
16 Chairman. I take it then that the Board's direction to
17 our clients is that our case will start no sooner than
18 the Tuesday of the second week in January?

19 THE CHAIRMAN: That's correct. And you,
20 of course, will be serving your witness statements
21 within the--

22 MS. CRONK: Calculated time.

23 THE CHAIRMAN: --calculated time based on
24 that; is that correct?

25 MS. CRONK: Yes. Thank you, sir. Thank

1 you very much.

2 THE CHAIRMAN: Okay.

3 PETER KINGSBURY,
4 LEONARD RITTER, Resumed

5 CONTINUED CROSS-EXAMINATION BY MS. CRONK:

6 Q. Dr. Ritter, I wonder if we might
7 continue from where we were yesterday. You had before
8 you when we were discussing Exhibit 716 an extract from
9 the Crump risk assessment analysis on the aerial -- the
10 risks of aerial spraying of pesticides.

11 We had a discussion yesterday towards the
12 end of the day about the approach that had been used by
13 the authors of this report and, to a certain extent,
14 the methodology, you recall that at the end of the day
15 yesterday?

16 DR. RITTER: A. Yes.

17 Q. Am I correct, Dr. Ritter, that the
18 approach and the methodology to risk assessment adopted
19 by the authors, as you described it yesterday, is one
20 which would only overestimate risks and could not
21 underestimate risks?

22 A. I think I would substitute your word
23 only for most probably.

24 Q. All right. Thank you. Could I ask
25 you then, if you would please, to turn back to page

1 (iv) of the executive summary that we were looking at
2 yesterday. I understand what follows after the end of
3 the discussion on page (iv) to be two summary tables
4 and I would like to review those with you.

5 Just dealing with the first table, Table
6 1, which appears over on the next page, page (v), there
7 is a description of the nature of the data contained in
8 that table set out at the bottom of page (iv) and could
9 I direct you to the last paragraph, the first part of
10 which reads, the bottom of page (iv):

11 "The highest estimated risks from
12 occupational exposures typically were for
13 loaders whose job responsibility involves
14 moving the herbicide from its container
15 to the mix tanks and from there to the
16 helicopter."

17 So stopping there for a moment, that
18 means, as I understand it, that of all of the risks
19 identified in the risk analysis for persons
20 occupationally exposed to pesticides, those with the
21 highest risks were loaders?

22 A. Yes, I read that the same way.

23 Q. All right. And then continuing with
24 the next sentence:

25 "The following table..."

1 Referring to Table 1 on the next page:

2 "Gives margins of safety from worst-case
3 estimates of exposure and worst-case
4 estimates of lifetime risk of cancer to
5 loaders from a single spray operation."

6 Stopping there. Do I understand that
7 correctly, Dr. Ritter, to mean that what is set out in
8 the table on the next page, first, is specific to the
9 highest risk group; that is, loaders and; secondly,
10 that the margins of safety are for the two worst-case
11 situations; that is, the worst-case exposure and the
12 worst-case estimates of lifetime risk of cancer?

13 A. I would read that the some way.

14 Q. All right. It's the worst on both
15 accounts?

16 A. Yes.

17 Q. All right. Then finally in the same
18 paragraph the authors continue to indicate:

19 "These expressions of risk represent the
20 size of the population actually exposed
21 according to the specific scenario, not
22 the total population potentially exposed.
23 For example, an estimated extra lifetime
24 cancer risk of one in one million does
25 not represent any population of one

1 million persons but rather one million
2 persons actually exposed to that amount
3 of specific herbicide under the specified
4 exposure scenario."

5 Now, stopping there and dealing with what
6 the authors are indicating. First, the extra lifetime
7 cancer risk is the concept that you discussed with the
8 Board yesterday?

9 A. That's correct.

10 Q. That is the approach taken in risk
11 analysis of carcinogenic risk?

12 A. Yes, it is.

13 Q. And what significance do you attach
14 to the indication that the authors have given that the
15 population -- the expression of one in one million,
16 represents those actually exposed to a specific
17 pesticide as opposed to those potentially to be
18 exposed?

19 What significance should we attach to
20 that, if any? What do they say?

21 A. It is somewhat of a departure from
22 the convention in doing these kinds of calculations,
23 but I would think it would, again, have the tendency of
24 exaggerating the risk estimate.

25 Q. What does it mean, though?

1 A. It means that in the calculation one
2 has presumed that all of those people are exposed
3 rather than simply potentially exposed, and that if
4 they were all exposed this would be the excess risk.
5 But if there is no assumption that there is any
6 possibility of it, they might not be exposed.

7 Q. All right. Does that mean, for
8 example then, before we turn to the actual numbers,
9 that if the risk -- the quantification of the risk
10 resulted in a number, for example, of .06, just for
11 example, per million that that would mean that of one
12 million persons actually exposed to cancer only six had
13 an extra risk of actually suffering carcinogenic
14 effects?

15 A. Not quite. It means if one million
16 persons actually exposed, the excess risk in that group
17 of one million would be .6 additional cancers, which is
18 not quite what you said but similar to it.

19 Q. All right. Can we look then at the
20 actual data and perhaps you can explain it further when
21 we look at the numbers.

22 Table 1 on the next page, as I understand
23 it, first of all, is the table setting out the
24 worst-case for occupational exposures. This is the one
25 dealing with loaders under the two worst-case

1 situations; is that correct?

2 A. Yes, it is.

3 Q. All right. And it deals both with
4 non-carcinogenic risks and as well cancer risks?

5 A. Yes.

6 Q. All right. Could we look first at
7 the non-carcinogenic risks. Am I correct that -- let's
8 just look at the column so I am sure that we are
9 understanding one another as we move through these
10 tables. The first column identifies the nature of the
11 herbicide?

12 A. Yes.

13 Q. The next three columns, I suggest,
14 entitled -- with a caption above it entitled: Margins
15 of Safety, pertain to non-carcinogenic effects?

16 A. That's correct.

17 Q. The last column deals with, as
18 stated, cancer risks?

19 A. Yes.

20 Q. All right. Could we deal first then
21 with the non-carcinogenic risks as set out in the
22 table.

23 Would you agree with me -- and I am
24 concerned only with 2,4-D and glyphosate, Dr. Ritter,
25 those being the herbicides authorized for aerial spray

1 use in forestry in Ontario, so I am just looking at the
2 first two chemicals.

3 For those risks identified for
4 reproductive and teratogenic effects, would you agree
5 with me that the margins of safety are all high for
6 2,4-D and glyphosate; they range from 137 to over a
7 thousand?

8 A. I prefer your second suggestion, they
9 range from 137 to over a thousand. As to whether or
10 not they are high, I think is a matter of judgment.

11 Q. All right. The risk of the effect,
12 would you agree, even for loaders, assuming these two
13 worst-case assumptions; that is, the worse estimate of
14 exposure, is low?

15 A. Yes.

16 Q. All right. And the margins of safety
17 for systemic effects are lower than they are for
18 reproductive and teratogenic effects, but they are at
19 least five for 2,4-D and 139 for glyphosate?

20 A. That's correct, yes.

21 Q. And if we look at cancer risks, they
22 are expressed per million?

23 A. Yes.

24 Q. Would you agree with me that the
25 risks are negligible for both 2,4-D and glyphosate?

1 A. Yes.

2 Q. .04 for 2,4-D -- well, it ranges from
3 .04 for 2,4-D to 9 per trillion for glyphosate; is that
4 correct?

5 A. Yes.

6 Q. Could we turn now to the worst-case
7 for environmental exposures as opposed to occupational
8 exposures.

9 If I can stop here for a moment just to
10 be clear. As I understand what the authors of this
11 report did, they looked at a variety of potential risk
12 situations or scenarios depending on, in the
13 occupational context, the job function?

14 A. That's correct.

15 Q. Right. And in the environmental
16 context that refers; does it not, to bystander or
17 general public exposure risks?

18 A. That's right.

19 Q. And, again, the authors looked at a
20 variety of potential risk situations under various
21 exposure scenarios; is that correct?

22 A. That's correct.

23 Q. So that when we look at Table 1, as
24 we just have with respect to occupational risk, what
25 they are saying is of everything that we looked at for

1 those persons whose occupations may expose them to
2 2,4-D and glyphosate the category of person with the
3 highest exposure -- with the highest risk were loaders
4 and these are the results even for them?

5 A. That's right.

6 Q. All right. And then when we come to
7 the environmental exposures, the summary table, Table 2
8 over on the next page deals again not with all of the
9 scenarios that the authors considered in their
10 analysis, but rather with the worst-case?

11 A. That's correct.

12 Q. Right. And if we -- could I direct
13 you first to the bottom of page (v) which is the
14 explanatory text for Table 2, being the summary table
15 for environmental risks. The authors indicate:

16 "The scenario which gave the highest
17 worst-case estimates of environmental
18 exposures was eating 20 helpings of
19 unwashed berries picked directly after a
20 spray operation. The following table
21 gives margins of safety from worst-case
22 estimates of exposure and worst-case
23 estimates of lifetime risk of cancer from
24 eating berries."

25 Stopping there. Again, Dr. Ritter, if I

1 could seek your assistance.

2 As I understand it then, what the authors
3 are indicating is that of all the risk scenarios in the
4 environmental exposure category that were looked at,
5 those which quantifiably had the highest degree of risk
6 were for persons who ate 20 helpings of unwashed
7 berries directly after a spray operation; is that
8 correct?

9 A. I would read that the same way, yes.

10 Q. All right. And, once again, in
11 setting out the summary data results they have taken
12 the same approach as they did for the highest risk
13 category of occupational exposures. They have
14 indicated what the risk assessments were for those who
15 had the worst exposures; that is, the most exposure,
16 and the worst-case estimates of lifetime risk of
17 cancer; is that correct?

18 A. Yes.

19 Q. All right. Could we go then directly
20 to the table and could we deal again, first, with the
21 non-carcinogenic effects and the risk of that. For
22 2,4-D and glyphosate they range from 23 to over 2,200;
23 am I right?

24 A. Yes.

25 Q. They are all larger than the margins

1 of safety for occupational exposures that we just
2 looked at in Table 1?

3 A. Yes.

4 Q. Does this mean, Dr. Ritter, that
5 there is less chance of the health effects referred to
6 in Table 2 occurring as a result of eating unwashed
7 berries than from working as a loader in spray
8 operations? Am I reading it fairly?

9 A. Yes.

10 Q. All right. And the margins of
11 safety, I suggest, given that they range from 23 to
12 over 2,000 are again very healthy; are they not?
13 Perhaps that's a bad choice of words in the context.

14 A. They are larger than they are for
15 occupational exposure.

16 Q. The risks of these effects actually
17 being sustained is very low?

18 A. That's right.

19 Q. All right. In fact some might say
20 negligible; would you agree?

21 A. Not necessarily. I think before one
22 can attach the term negligible particularly to things
23 like systemic effects it is important to understand
24 what the nature of the effect is.

25 The margin of safety in itself when

1 quoted as an 'absolute number doesn't have a great deal
2 of meaning until one understands the particular end
3 point to which that number is being modeled.

4 In other words, reproductive effects or
5 teratogenic effects in itself is not very informative.
6 There are many variables in a teratology study that are
7 evaluated, some of significance and others which are
8 relatively trivial.

9 Q. Can we fairly take, however, from
10 this quantification - and let's take, for example, the
11 teratogenic effects category - can we fairly take from
12 it that it was the result of the analysis that
13 potentially human health effects dealing with fertility
14 or miscarriage were low?

15 A. Yes.

16 Q. All right. And similarly with
17 respect to the reproductive effect category, such
18 matters as birth -- risk of birth defects or
19 malformations resulting from exposure to these two
20 chemcials, those risks were low?

21 A. Yes.

22 Q. All right. Can we look then at the
23 carcinogenic risk, the cancer risk on the right-hand
24 side. They range, if I am reading the table correctly,
25 from 9 per billion for glyphosate to 19 per million for

1 2,4-D. Am I reading it correctly?

2 A. That's right.

3 Q. All right. They are, I suggest, a
4 very low negligible risk?

5 A. I'm sorry, I think you said 9 per
6 billion, I think that's actually .9 per billion.

7 Q. Sorry, all right.

8 A. It is less than one per billion.

9 Q. I am sorry. So it's .9--

10 A. Yes.

11 Q. --per billion?

12 A. Yes.

13 Q. And is it to 19 per million, or is it
14 .19?

15 A. No, it is .19.

16 Q. All right.

17 A. Or if you like 19 per billion rather
18 than per million.

19 Q. All right. I suggest, Dr. Ritter,
20 that those risks are indeed negligible; would you
21 agree?

22 A. Yes, I would.

23 Q. All right.

24 THE CHAIRMAN: Dr. Ritter, how do you
25 reconcile the statement above the tables saying:

1 "Those herbicides indicated by an
2 asterisk have not been shown to be
3 carcinogens."

4 And 2,4-D does not have an asterisk with
5 the other study, the Ontario study, Exhibit 714?

6 DR. RITTER: Which indicated that 2,4-D
7 was held not to be a carcinogen.

8 THE CHAIRMAN: Right.

9 DR. RITTER: I think you will find, Mr.
10 Chairman, that that's on the basis of the dates during
11 which these analyses were done. The Krump analysis was
12 submitted in May of 1986 and the bulk of the work which
13 went into this was actually conducted between 1984 and
14 1985.

15 At that time the information that was
16 available on 2,4-D may have led some to conclude, at
17 least in the absence of some clarification that came
18 later on, that it was reasonable to assume that it was
19 at least an animal carcinogen. Subsequent to that
20 there were some additional analyses and investigations
21 that were done on 2,4-D, and at about the time that the
22 Ontario people were looking at it, I think most sources
23 felt that the evidence was no longer sufficiently
24 convincing to arrive at that conclusion.

25 THE CHAIRMAN: So if this were done

1 today, Exhibit 716 study, it would have an asterisk in
2 front of it?

3 DR. RITTER: I think it probably would,
4 yes.

5 THE CHAIRMAN: Thank you.

6 MS. CRONK: Thank you.

7 Q. I would like to come back to that
8 answer you have given, Dr. Ritter, in the context of
9 the World Health Organization report on 2,4-D that was
10 done that you mentioned yesterday, but can you confirm
11 to the Board that during the years 1984 to 1987 there
12 was a considerable amount of continuing research done
13 on the issue of the possible carcinogenic effect of
14 2,4-D?

15 DR. RITTER: A. Yes.

16 Q. All right. And so that a report
17 written in 1984 based on the information database that
18 existed at that time might, when viewed in today's
19 light, not reflect the consensus of scientific
20 community position on the issue that would pertain
21 today?

22 A. Yes, I would agree with that. The
23 two pivotal cancer studies I believe only became
24 available in 1986 which would have been after the time
25 that the Crump report was written.

1 Q. But before the time they were
2 considered in the MOE report; were they not?

3 A. That's right. The MOE report would
4 have considered and did consider in fact the two most
5 recent cancer studies. The Crump Report I don't think
6 could have.

7 Q. All right. And we will come to it
8 later this morning, but with respect to the World
9 Health Organization Report that you spoke about
10 yesterday, first, that is specific to 2,4-D; is it not?

11 A. Yes, it is.

12 Q. Secondly, it was published in 1984?

13 A. Yes.

14 Q. And has, therefore, the same
15 limitation that you have just mentioned with respect to
16 the Crump Report?

17 A. Yes, it does.

18 Q. Thank you. Then if we could, just
19 dealing with the cancer risk column for Table 2 before
20 we leave it, the authors have attempted to put, as they
21 describe it, those risks or those chances of cancer in
22 perspective and they do so in the last sentence on page
23 (vi) where they indicate:

24 "In order to put these estimates into
25 perspective we note that the cancer risk

1 from smoking a single cigarette is about
2 0.6 per million; that from spending a day
3 in the Rocky Mountains at an altitude of
4 10,000 feet from the extra radiation
5 both cosmic and terrestrial is about
6 0.13 per million, and the extra risk of a
7 fatal automobile accident from not
8 wearing a seat belt for one day is about
9 0.3 per million."

10 Now, stopping there, Dr. Ritter, those
11 are all activities which are not infrequently engaged
12 in by some sectors of the general public; you would
13 agree?

14 A. Yes.

15 Q. In fact they might even be engaged in
16 in concert?

17 A. Yes.

18 Q. It seems, based on these examples -
19 and I don't mean to be facetious in any way by this -
20 that the highest risk of common, as an example, of
21 common activities engaged in by the general public
22 would be driving an automobile in the Rocky Mountains
23 without your seat belt on and having a cigarette. You
24 would agree?

25 A. Yes.

1 Q. All right.

2 THE CHAIRMAN: And hitting a truck.

3 MS. CRONK: I'm sorry?

4 THE CHAIRMAN: And hitting a truck.

5 MR. CRONK: Yes, and hitting a truck,
6 that's right. That would take it perhaps off the map
7 in terms of the degree of risk.

8 Q. But my point, in a serious way, is
9 simply this, Dr. Ritter, that when we look at the
10 degree of risk quantified in this study for cancer
11 risks for either 2,4-D or glyphosate, they are, as you
12 already indicated or agreed with me, very low,
13 negligible, but in the context of risks to which
14 members of the general public are routinely exposed
15 they seem indeed very negligible; would you agree?

16 A. Yes.

17 Q. Thank you. Now, in both Table 1 and
18 Table 2 the summary results for the worst-case risk of
19 human health effects for picloram is also set out, but
20 I should simply indicate to the Board and perhaps you
21 can confirm in two parts:

22 First, the Board has received evidence
23 and the evidence is that picloram is not authorized for
24 aerial spraying in Ontario. So I ask you simply to
25 accept that. Am I correct that this study was based on

1 aerial spraying conditions?

2 A. Yes, it was.

3 Q. All right.

4 MS. CRONK: And it is for that purpose,
5 Mr. Chairman, that I won't be directing questions to
6 the witness on the picloram results.

7 Q. Could I ask you to go then -- I would
8 like to spend just a few moments, if we could, on the
9 actual detailed results for both glyphosate and 2,4-D
10 which are included in the extract forming this exhibit,
11 and could we deal first with glyphosate, please.

12 There are four tables set out at pages
13 153 to 157 and they, as I understand it, are the
14 depiction in tabular form of the results of the risk
15 assessment for glyphosate; is that correct?

16 DR. RITTER: A. Yes.

17 Q. All right. The first three tables
18 beginning at page 153, Table 5-5 and the next two
19 tables deal with non-carcinogenic effects; is that
20 correct?

21 A. That's correct.

22 Q. The fourth deals with cancer risks?

23 A. Yes.

24 Q. All right. So if we could again
25 start with non-carcinogenic effects, Table 5.5, as I

1 understand it, deals with and sets out the margins of
2 safety for non-carcinogenic systematic effects from
3 exposure to glyphosate through a single spraying
4 episode; correct?

5 A. That's correct.

6 Q. All right. Now, just stopping there
7 for a moment. This table is constructed in somewhat of
8 a different fashion from the summary tables that we
9 just looked at and I would ask, if you could, simply to
10 recall the discussion we had yesterday about the
11 contents of the executive summary indicating the way
12 the authors had approached this risk analysis.

13 Am I correct that what's set out in Table
14 5-5 are the quantification of risks which were the
15 result of the risk assessment for both the worst-case
16 risk scenario and, as well, what the authors termed to
17 be a more reasonable risk scenario?

18 A. Yes, it is.

19 Q. All right. Not as extreme, more
20 reasonable. All right.

21 Dealing then -- and that explains; does
22 it not, the data set out under the more reasonable
23 columns and the worst-case columns?

24 A. Yes.

25 Q. That's what they are?

1 A. Yes. .

2 Q. And under each column the estimated
3 human exposure is indicated and that is expressed in
4 micrograms per kilogram of body weight per day?

5 A. That's correct.

6 Q. And then the margin of safety is
7 indicated directly beside it?

8 A. Yes.

9 Q. And the risks quantified for both
10 occupational exposures and environmental exposures are
11 set out?

12 A. Yes.

13 Q. All right. Environmental again being
14 bystander or general public exposure?

15 A. Yes.

16 Q. All right. Dealing then, first with
17 the results for systematic effects, am I correct that
18 the margins of safety under both the reasonable and the
19 worst-case scenarios for glyphosate range from 138
20 through to 5,000?

21 A. Yes.

22 Q. Again, would you agree with me that
23 the risks, based on those quantifications of actually
24 suffering systematic effects, is low?

25 A. Yes.

1 Q. And if we look at what the authors
2 have termed the more reasonable actual risk exposure
3 situations, the margins of safety range from 555 to
4 5,000?

5 A. Yes.

6 Q. All right. And that's for
7 occupational exposures. When we move into the
8 environmental category, I suggest to you that the
9 margins of safety are much, much higher and that,
10 therefore, the degree of risk is much, much lower?

11 A. That's correct.

12 Q. And in fact, in some instances, we
13 are talking about a margin of safety of 550,000?

14 A. That's correct.

15 Q. Could I ask you then to go to Table
16 5-6. Perhaps we could, as the format has now been
17 discussed, go through these rather quickly. This table
18 deals; does it not, with non-carcinogenic potential
19 reproductive effects?

20 A. Yes, it does.

21 Q. Again, I suggest to you that in both
22 the reasonable and the worst-case situations for both
23 those occupationally exposed and those environmentally
24 exposed the margins of safety are quite high?

25 A. Yes.

1 Q. In some cases extremely high?

2 A. Yes.

3 Q. The risk, therefore, of actually

4 suffering these effects is low?

5 A. Yes.

6 Q. Table 5-7 dealing with

7 non-carcinogenic teratogenic effects. Again, margins

8 of safety in both the reasonable and the worst-case

9 scenarios are very high; in some cases, extremely high?

10 A. Yes.

11 Q. Again the degree of risk, therefore,

12 is very low?

13 A. Yes.

14 Q. And finally, if we could go to Table

15 5-8 dealing with cancer risks. On page 156, in this

16 instance the quantification of risk is expressed

17 differently and, as you explained yesterday, does not

18 involve margins of safety per se?

19 A. That's correct.

20 Q. Is that correct? And as well, the

21 mathematics are expressed in a different format than we

22 have seen in the previous tables?

23 A. That's right.

24 Q. All right. Am I correct, however,

25 that in looking at the quantification of risk; that is,

1 risk of cancer from exposure to glyphosate both in the
2 occupational exposure context and in the environmental
3 exposure context and both in the reasonable and
4 worst-case scenarios, that the risks of cancer are very
5 low indeed?

6 A. Yes.

7 Q. In fact negligible?

8 A. Yes.

9 Q. And could I take you to the comments
10 of the authors regarding these tables at page 140
11 section under 5.3 dealing with risk characterization
12 and this pertains, Dr. Ritter, to glyphosate.

13 The beginning of -- beginning with the
14 second full paragraph on that page, do we find there
15 the authors' comments on the results set out in the
16 following tables that we have just reviewed?

17 A. Yes.

18 Q. And dealing first with the
19 teratogenic effects indicated in Table 5-7 the authors
20 suggest -- well, perhaps I should read it to you in
21 fairness:

22 "All estimated margins of safety reported
23 in Table 5-7 exceed 1,000 even in the
24 worst-case, implying that the risk of
25 teratogenic effects from exposure to

1 glyphosate through a single spraying
2 episode is negligible if interpreted in
3 terms of the traditional one hundred fold
4 safety factor."

5 Do you agree with that interpretation of
6 the results?

7 A. I agree with it, but in fact I think
8 it's somewhat misleading. Glyphosate is actually not
9 considered to be a teratogen.

10 Q. All right.

11 A. So that the statement here that
12 implies that there is greater than a one thousand fold
13 margin of safety for teratogenic effects leaves one
14 with the impression that there are teratogenic effects
15 to which this one thousand fold margin of safety
16 exists.

17 Q. Right.

18 A. As I recall, that is not correct.
19 The one thousand fold margin of safety is to a
20 teratology study but not to teratogen effects per se.

21 The effects in that study which are being
22 using to margin this model of safety are not based on
23 birth defects, but rather to effects in the teratology
24 study which I would certainly consider to be much less
25 important than, frankly, birth defects.

1 Q. All right. Well, thank you for that
2 clarification then. And dealing with the authors'
3 comments concerning the results pertaining to
4 systematic or reproductive effects in the next two
5 sentences of that paragraph, the authors suggest, and
6 they describe, first of all, what the actual results
7 were and they suggest that these results indicate that:

8 "With respect to non-carcinogenic risk
9 the primary cause for concern, if any,
10 from exposure to glyphosate is related to
11 possible systematic effects. It should
12 be recognized, however, that these
13 margins of safety are conservative in the
14 sense that they compare a single-day
15 human exposures to average daily animal
16 exposures over 90 days. In the
17 worst-case scenario a worker would have
18 to encounter 139 times the worst-case
19 single-day exposure to reach a level
20 equal to the NOEL, only above which are
21 adverse effects considered to be likely."

22 They go on to indicate that:

23 "A margin of safety of 298 corresponding
24 to the worst-case environmental scenario
25 for exposure to glyphosate and that

1 298..." sorry.

2 "A margin of safety of 298 corresponding
3 to the worst-case environmental scenario
4 for exposure to glyphosate from eating
5 wild berries means that an individual
6 would have to consume approximately 160
7 pounds of contaminated wild berries in
8 one day in order to reach an exposure
9 equivalent to the NOEL for glyphosate."

10 Now, just dealing with those comments,
11 Dr. Ritter. First, the conservatism that the authors
12 suggest is that which we discussed yesterday; namely,
13 that they have, for the purposes of their analysis,
14 compared single-day human exposures to average daily
15 animal exposures over 90 days?

16 A. That's correct.

17 Q. Correct. And would you agree with
18 the implications of the authors' remarks that the
19 degree of risk, when viewed in that perspective, is
20 indeed quite low?

21 A. I would agree that when viewed in
22 that perspective the risk is quite low. Exposure may,
23 of course, in the human case take place for more than
24 one day.

25 Q. Yes.

1 A. So I think they have -- in my view,
2 they have somewhat over extended the value of that
3 conservatism, if you like.

4 Q. All right. And I will come back with
5 respect to all of these to the general issue of repeat
6 exposure.

7 And the authors' discussion with respect
8 to the significance of the cancer results for exposure
9 to glyphosate are also set out at page 141 in the last
10 two paragraphs. And, again, they indicate what the
11 actual results were and they suggest that the estimated
12 risk from occupational exposure to glyphosate through a
13 single spraying episode range from 9 in 100-billion to
14 1 in 100-billion?

15 A. Yes.

16 Q. All right. And they further indicate
17 and conclude on the basis of their analysis that the
18 results suggest that the carcinogenic risk from
19 exposure to glyphosate through a single spraying
20 episode is negligible both for occupationally exposed
21 and for environmentally exposed individuals?

22 A. Yes.

23 Q. Would you agree with that?

24 A. Yes, I would.

25 Q. All right. Could we look then next,

1 if we could please, at the results for 2,4-D and
2 perhaps we will just do it from the back forward, so
3 that we will deal first with the cancer risks, that is
4 Table 4-8 at page 130.

5 And would you agree with me, looking at
6 the risk assessment results for cancer risks as set out
7 in this table, again both in the occupational and
8 environmental exposure categories, and both under the
9 more reasonable and the worst-case scenarios, that the
10 results indicate negligible risks of cancer for persons
11 in either category under either scenario?

12 A. Yes.

13 Q. And dealing with the teratogenic
14 effects they are set out in Table 4-7. Would you agree
15 with me that the results reported indicate large
16 margins of safety under all the reasonable scenario
17 situations and under most of the worst-case scenarios?

18 A. Yes.

19 Q. And extremely high margins of safety
20 in many cases?

21 A. Yes.

22 Q. Dealing with the non -- suggesting
23 that the risks of exposure -- the risks of sustaining
24 effects of that kind are low?

25 A. Yes.

1 Q. And dealing with the non-carcinogenic
2 reproductive effects, Table 4-5, there are similar
3 results, similar -- I mean similar in the context to
4 the ones that we have just considered for teratogenic
5 effects; namely, there are very high margins of safety
6 again?

7 A. You are referring to Table 4-6?

8 Q. Yes, I was. I beg your pardon.

9 A. Yes.

10 Q. And indeed in many cases they are
11 very high; are they not?

12 A. Yes.

13 Q. Over 2-million in some instances?

14 A. Yes.

15 Q. Closer to 3-million in fact?

16 A. Yes.

17 Q. And then finally, dealing with Table
18 4-5, systematic effects, would you agree with me that
19 the margins of safety for systematic effects are lower
20 than with respect to the margins of safety for other
21 non-carcinogenic effects?

22 A. Yes.

23 Q. And they are lowest under obviously
24 the worst-case scenario?

25 A. Yes.

1 Q. And within that scenario they are the
2 lowest for a loader where the margin of safety is 5.49?

3 A. That's correct.

4 Q. And that is the worst-case highest
5 risk category dealt with in the summary table, Table 1,
6 that we looked at?

7 A. Yes.

8 Q. All right. And the highest is
9 110,000 for ingestion of 2,4-D affected fish. Is that
10 correct?

11 A. That's correct.

12 Q. Would you agree with me as well that
13 in many cases the margin of safety is very high under
14 both the worst-case and the more reasonable case
15 scenario?

16 A. For environmental?

17 Q. Yes.

18 A. Yes.

19 Q. Yes. And even in the case of the
20 lowest margin of safety, as the authors indicated, a
21 worker would have to encounter 5.5 times the single-day
22 exposure to reach a level equal to the NOEL; isn't that
23 so?

24 A. That is what that says, yes.

25 Q. All right. Then dealing generally

1 with the implications of this study, Dr. Ritter, and
2 bearing in mind what you told the Board both about the
3 expertise and experience of Dr. Crump in this field and
4 the nature of the available information in 1985 and '86
5 when this report was prepared, would you agree with me
6 that based on this risk assessment report, in practical
7 terms, there is negligible risk to humans of cancer
8 afforded by exposure to 2,4-D or glyphosate?

9 A. Yes, I would.

10 Q. Would you also agree with me that in
11 terms of non-carcinogenic effects this report again
12 substantiates the view that there is negligible risk to
13 humans of non-carcinogenic health effects by exposure
14 to 2,4-D or glyphosate?

15 A. I think one might argue as to whether
16 or not a margin of safety of six or five fold
17 constitutes a negligible risk. With that qualification
18 I would agree with you, yes.

19 Q. All right. And that qualification
20 pertains to possible systematic effects for exposure to
21 2,4-D only?

22 A. That's right. But as I indicated,
23 it's difficult to attach a value judgment to the
24 significance of that margin of safety without an
25 understanding of the end point to which that margin of

1 safety has been modelled.

2 Q. Do you have any concerns, based on
3 the results in this report, Dr. Ritter, that there is a
4 significant risk of human health -- negative human
5 health effect as a result of exposure to 2,4-D or
6 glyphosate?

7 A. No.

8 Q. Thank you. In either the
9 occupational or the environmental exposure categories?

10 A. That's correct.

11 Q. That's correct, meaning you do not?

12 A. That's right.

13 Q. All right. And I said I would come
14 back to the issue. Do you regard this report then as
15 being reliable?

16 A. Yes, I do.

17 Q. All right. Could I ask you then to
18 go back to the issue of more than single-day exposure
19 and, as I said I would return to, and in that regard
20 all of the tables that we have looked at pertain; do
21 they not, to single-spray operations?

22 A. Yes.

23 Q. In terms of quantifying the degree of
24 human risks?

25 A. That's right.

1 Q. However in arriving at the margin of
2 safety, for example, in the non-carcinogenic effect
3 category, single-day exposure for humans was compared,
4 to arrive at the margin of safety, with multiple
5 exposure situations based on animal tests; is that
6 correct? 90-day exposure.

7 A. Not exclusively. Some of these end
8 points that have been modelled are to teratology and
9 reproduction and the principle of repeat dosing.

10 The teratology study, for example, is
11 relatively meaningless. I can -- I don't know if that
12 is worth pursuing or not, but the window of exposure
13 necessary to induce a birth defect either in humans or
14 experimentally is --

15 Q. Narrow.

16 A. --is relatively narrow.

17 Q. Yes.

18 A. Now, in the case of animal studies,
19 that window is expanded somewhat to ensure that the
20 precise moment in time when that birth defect can be
21 induced is covered, so that one typically will cover a
22 rather significant proportion of the gestational period
23 just to cover off that possibility.

24 In reality we know that it takes a much
25 narrower window of exposure. So that the principle of

1 the repeat exposure in something like a teratology
2 study, for example, is relatively meaningless because
3 it is not repeat exposure that is necessary to produce
4 the effect, it was typically only a single day's
5 exposure.

6 So, in that case, a single day's exposure
7 is more directly referable to the nature of the
8 protocol in the study than it would be, for example, in
9 a 90-day study.

10 Q. All right. Am I correct, however,
11 that where 90-day studies were done in accordance with
12 appropriate protocol, it is the results under those
13 90-day studies that formed one of the benchmarks to
14 arrive at the margin of safety for human exposure?

15 A. That's correct.

16 Q. All right. And I think I understand
17 what you are saying with respect to the teratogenic
18 studies, for example, simply being that there were some
19 where there wasn't a 90-day situation because that
20 wouldn't have been an appropriate way to proceed with
21 testing in any event?

22 A. What I'm really saying is that the
23 teratology study should really be seen as a one-day
24 exposure study. Although exposure may have taken place
25 for 10, or 11 or 12, it was only single day's exposure

1 that produced whatever effect has been seen.

2 Q. I understand. All right. Now,
3 having said that, could I ask you to go, if you would,
4 back to the text of the executive summary and, in
5 particular, to page (viii).

6 And I direct your attention, Dr. Ritter,
7 particularly to the second paragraph which deals, as I
8 understand it, with the possibility of multiple
9 exposures and the authors indicate:

10 "Synergism (antagonism) is said to occur
11 when the effects from exposure to
12 multiple chemicals are greater/less than
13 expected."

14 Now, stopping there for a moment, can you
15 distinguish for me, in this context, between synergism
16 and antagonism; or are they the same?

17 A. They are not the same. Synergism is
18 where the effects of two different chemicals given
19 simultaneously would be the greater than the sum of
20 either chemical alone. Antagonism is simply the
21 converse of that.

22 Q. All right. So we are not talking
23 about multiple exposure, we are talking about different
24 compounds in that regard?

25 A. That's right.

1 Q. Then the authors continue:

2 "We feel that biologically significant
3 synergistic or antagonistic responses are
4 unlikely under the given exposure
5 conditions for the herbicides being
6 studied because exposures to different
7 herbicides will generally be separated in
8 time and because risks from exposure to
9 each herbicide individually are estimated
10 to be very small."

11 Now, stopping there for a moment. You
12 might not be aware, Dr. Ritter, but this issue of
13 possible synergistic effect has arisen through the
14 course of other evidence before the Board.

15 Do you agree or disagree with the
16 conclusion suggested by the authors in the sentence I
17 have just read to you?

18 A. I neither agree nor disagree because
19 I am not aware of the use, I am not that personally
20 familiar with the use practice in Ontario.

21 If you are telling me that these
22 chemicals are not used temporally, close enough in time
23 so that one can really view it as a possible
24 synergistic effect, then I'm prepared to take that at
25 face value.

1 Q. All right. Would you agree with me
2 that based on the risk analysis in this report,
3 however, set aside the issue of actual use pattern in
4 Ontario--

5 A. Mm-hmm.

6 Q. --and dealing just with the
7 quantification of risks in this report, that the risks
8 are in fact estimated in this report to be very small--

9 A. Yes.

10 Q. --based on the exposures dealt with
11 in this report?

12 A. Yes.

13 Q. All right. And then, finally, the
14 authors indicate:

15 "Consequently, risks from multiple
16 exposures were estimated by assuming that
17 Synergism or antagonism do not occur;
18 i.e., by simply summing the risks from
19 individual exposures."

20 A. Yes.

21 Q. Now, can you help me as to what that
22 means in the context of the data we have looked at?

23 A. What the authors have done here is
24 where there has been a calculation of multiple
25 exposure, they have assumed that exposure has taken

1 place on several days to one chemical alone rather than
2 to a series of chemicals.

3 Q. Thank you.

4 MRS. KOVEN: Excuse me, Dr. Ritter.
5 Would they have also looked at a worker exposure and
6 residential exposure and recreational exposure in that
7 summing?

8 DR. RITTER: They may have. I can't
9 really tell you precisely for any given instance
10 immediately if they did or they did not, but if one
11 were to look at environmental exposures, for example
12 for most of the risks that have been investigated here,
13 they are sufficiently small and I think it is very
14 unlikely that they would make an important contribution
15 to the overall calculation of risk.

16 When one sums a series of very small
17 numbers, one still ends up with a small number.

18 MS. CRONK: Mrs. Koven, could I just ask
19 you to repeat the three categories that you mentioned.
20 It was worker exposure, residential and, I'm sorry, I
21 missed the third.

22 MRS. KOVEN: And a recreational exposure.

23 MS. CRONK: Recreational. Thank you.

24 MRS. KOVEN: You know, the situation in
25 northern Ontario in terms of use pattern is that there

1 are probably multiple sources of exposure, albeit
2 small, one would think that the summing, if you were
3 looking only in that northern Ontario experience - and
4 the risk assessment wasn't done that way - that that
5 would properly be part of it, I would think.

6 MR. RYDER: I think you are probably
7 correct. I think to be absolutely precise one would do
8 it that way, but on brief examination of the risks, as
9 I have indicated, on brief examination of the risks or
10 the exposures, whichever number one prefers, I would
11 think it is unlikely to significantly alter the overall
12 estimate because overall the estimates of exposure are
13 extremely small.

14 MS. CRONK: Q. Thank you, Dr. Ritter. I
15 would like to turn then very briefly to the World
16 Health Organization study concerning 2,4-D that you
17 mentioned yesterday.

18 Do you, by any chance, have a copy of it?

19 DR. RITTER: A. No, I don't.

20 Q. All right.

21 MS. CRONK: I should say, Mr. Chairman,
22 for the information of the Board I don't propose to
23 mark this but simply to refer very briefly to a number
24 of sections given that Dr. Ritter mentioned it. I made
25 you a copy. (handed)

1 THE CHAIRMAN: Thank you.

2 MS. CRONK: Q. Dr. Ritter, you have
3 already confirmed for me that this report was released
4 or available, as it were, in 1984; am I correct?

5 DR. RITTER: A. That's correct.

6 Q. And you indicated that it is
7 particular as the title suggests to 2,4-D?

8 A. That's right.

9 Q. And as I understand, it is a document
10 published under the joint sponsorship of the United
11 Nations Environment Program, the International Labour
12 Organization and the World Health Organization; is that
13 correct?

14 A. That's correct.

15 Q. And you indicated yesterday that your
16 laboratory was involved in the production of this
17 report; is that correct?

18 A. We were much more than simply
19 involved, we were requested by the World Health
20 Organization to convene an expert panel to write this
21 report and served as the secretariat for its
22 preparation.

23 Q. All right. And did you then have
24 responsibility for determining the list of experts
25 described in the report who participated in it?

1 A. Through some process of consultation,
2 yes.

3 Q. All right. And I think you have
4 already confirmed for me that the conclusions expressed
5 and the data reported upon in this document were based
6 on the state of scientific information available in
7 1984, and I suppose prior to that when the work was
8 being done?

9 A. That's correct.

10 Q. All right. Then, just dealing with
11 it at that point in time, because the report has come
12 up and it's been mentioned, I would like to refer you
13 to the section dealing with the evaluation of health
14 risks to man from exposure to 2,4-D; that is, Chapter 9
15 at page 99 very briefly, and I just ask for your
16 confirmation as to certain of the conclusions or
17 observations expressed in the report.

18 MS. CRONK: And I will simply put them on
19 the record, Mr. Chairman, unless there is any need to
20 have the pages marked.

21 Q. First, the first section in this
22 chapter; am I correct, deals with general
23 considerations relating to the evaluation of health
24 risks to man from exposure to 2,4-D?

25 DR. RITTER: A. That's correct.

1 Q. And am I correct that it suggests,
2 and I quote:

3 "In areas of 2,4-D herbicide production,
4 handling or use, the highest exposure
5 will be incurred by those who are
6 directly involved in these processes
7 followed by bystanders directly exposed
8 to 2,4-D vapour, dust or droplets or to
9 contaminated vegetation, soil, or water.
10 In these two groups, exposure will
11 usually be via the skin."

12 Now, stopping there for a moment. Do you
13 agree that for the bystander group the dermal exposure
14 is the significant feature -- factor?

15 A. It is a significant factor not only
16 for bystander but indeed for occupational.

17 Q. All right. So that when we look at
18 the tables from the Crump report in the environmental
19 risk category that we have looked at, those dealing
20 with exposure dermally become significant; do they not?

21 A. Absolutely.

22 Q. All right. And then again under
23 general considerations the text indicates:

24 "The general population in 2,4-D use
25 areas would be exposed to a lesser extent

1 mainly through food containing 2,4-D.
2 residues and to a lesser extent through
3 2,4-D residues in water. The
4 contribution from air is negligible. As
5 far as the general population is
6 concerned 2,4-D intake from any source is
7 negligible."

8 A. Yes.

9 Q. Do you agree with those observations,
10 Dr. Ritter?

11 A. Yes.

12 Q. All right. And then dealing with the
13 contents of the report regarding carcinogenic effects,
14 could I direct your attention to page 101, Section
15 9.3.2.5. Am I correct that the authors indicate
16 available animal bioassays and -- I'm sorry--

17 A. Epidemiological studies.

18 Q. Thank you. Are inadequate for an
19 assessment of the carcinogenic potential of 2,4-D or of
20 its derivatives. That is what the report indicates?

21 A. Yes.

22 Q. All right. Am I correct, Dr. Ritter,
23 that that was a conclusion clearly reached at the time
24 the report was prepared and not taking into account the
25 work that became available to the MOE expert panel and

1 indeed, at least in some part, to the Crump authors?

2 A. Yes.

3 Q. Thank you. Is that just another way
4 of saying -- is the conclusion or observation made in
5 this document, the World Health Organization report,
6 just another way of saying that the evidence -- the
7 then available scientific evidence was insufficient or
8 inadequate to conclude that 2,4-D was a carcinogen one
9 way or the other?

10 A. I would like to change that a little
11 bit, if I can. When one tests the null hypothesis;
12 that is, it's very difficult in science to test a
13 negative, so one can only test for the absence of a
14 positive effect; that is, the question one asks of a
15 cancer bioassay is: Did it cause cancer, rather than
16 the converse: Did it not.

17 Now, what this sentence says is that the
18 available information is inadequate to arrive at a
19 conclusion, rather than saying that the available
20 evidence did not suggest that it was a carcinogen.
21 Those are different conclusions.

22 Q. All right. I tried not -- All right.
23 I don't want to get into a semantical debate with you.
24 My point is, they couldn't tell based on the evidence
25 they had?

1 A. I'd agree with that.

2 Q. One way or the other?

3 A. That's right.

4 Q. All right. Which is, as you point
5 out, different what the Ministry of the Environment
6 expert panel said because they concluded in an
7 affirmative sense that the evidence was insufficient to
8 establish that it was carcinogenic?

9 A. That's correct. That's a very
10 different version.

11 Q. Exactly. Thank you. Then if we
12 could deal with the evaluation of health risks from
13 2,4-D exposure, section 9.4 page -- same page, am I
14 correct that the report indicates that from the data
15 available at present, the task group assumes that a
16 possible health risk will exist when the safety factor
17 is less than 100.

18 So just dealing with that, that is what
19 the authors of this report concluded as a possible
20 health risk standard or a benchmark, if I could put it
21 that way?

22 A. That's right.

23 Q. All right. Their recommendations on
24 exposure follow in the immediately following paragraph
25 and to paraphrase it, could I suggest that they

1 indicate that the risks of exposure can be reduced
2 fairly simply - that is their language - fairly simply
3 by measures of occupational hygiene, by proper
4 laundering procedures, et cetera?

5 A. Yes.

6 Q. All right. So that whatever the risk
7 might be, properly followed hygienic and protection
8 procedures can minimize that risk?

9 A. Yes.

10 Q. All right. And, indeed, that is
11 something that the -- well, in fairness to you I'll
12 take you to the section.

13 Could I also ask you to go back to
14 Chapter 1 which is a summary section of the document
15 and which deals with effects on human beings of
16 exposure to 2,4-D.

17 And I'm interested particularly in the
18 section at page 12, Dr. Ritter, dealing with the uptake
19 and fate of 2,4-D in the body and that is Section
20 1.1.5. Am I correct that the authors indicate that
21 2,4-D and its derivatives can be absorbed via the oral,
22 dermal and inhalation routes? Stopping there.

23 A. Yes.

24 Q. And that general population exposure
25 is mainly by the oral route but that under occupational

1 and bystander exposure conditions, the dermal route is
2 by far the most important?

3 A. Yes, that's --

4 Q. I'm sorry.

5 A. That's generally been true in most
6 investigations world-wide.

7 Q. All right. And that's just
8 confirmation of the passage that we looked at a few
9 moments ago?

10 A. Yes.

11 Q. All right. And then the point which
12 I'm particularly interested in at the moment, am I
13 correct that the authors indicate that there is no
14 evidence that 2,4-D is accumulated throughout the body?

15 A. That's correct.

16 Q. All right.

17 A. In fact I would say that, if I may,
18 that conclusion I would say has been strengthened
19 considerably since the time that this work was done.

20 Q. All right. And indeed, just dealing
21 with the state of information then available,
22 recognizing as you have said that it has been
23 strengthened today by the state of scientific
24 knowledge, but even then the authors indicated that:

25 "Transformation of 2,4-D in mammals

1 appears to occur only to a slight extent
2 and mainly involves the production of
3 2,4-D conjugates with sugars or amino
4 acids and that a single dose was excreted
5 within a few days."

6 A. That's what it says, yes.

7 Q. All right, thank you.

8 THE CHAIRMAN: Ms. Cronk, in view of the
9 number of passages you've read, I think it would be
10 advisable--

11 MS. CRONK: Fine, sir, I'll do it.

12 THE CHAIRMAN: --if we had copies of that
13 put into evidence.

14 MS. CRONK: I apologize for not having it
15 done. I read the report late last evening, but I'll
16 get them for you.

17 THE CHAIRMAN: Thank you.

18 MS. CRONK: Q. Can we agree then, Dr.
19 Ritter, and I would just like you to step back and
20 consider the four documents that we've talked about
21 over the last two days; first of all, the MOE expert
22 panel document, the Bond article dealing with phenoxy
23 herbicides generall, the Crump report, now the World
24 Health Organization Report that we've referred to.

25 Just comparing the World Health report,

1 for example, and the MOE expert panel report, would you
2 agree with me that the expert panel report prepared on
3 behalf of and delivered to the Minister of the
4 Environment in Ontario is clearly the more recent
5 document and contains strongly worded conclusions
6 regarding the possible carcinogenic effect of 2,4-D?

7 DR. RITTER: A. Yes.

8 Q. And, similarly, the World Health
9 Organization report predates the Crump report and does
10 not contain the same type of risk analysis data that is
11 contained in the Crump report?

12 A. That's correct.

13 Q. The purposes were different?

14 A. Yes.

15 Q. All right. And, again, the
16 conclusions expressed in the Crump report suggest
17 negligible or low risks of cancer or non-carcinogenic
18 health effects from exposure to 2,4-D and glyphosate
19 under the conditions they examined?

20 A. Yes, that report says that.

21 Q. All right. Are you aware of any
22 similar World Health Organization report regarding
23 glyphosate?

24 A. No, none has been done to the best of
25 my knowledge.

1 Q. All right. With respect to any of
2 the pesticides in use in Ontario, bearing in mind the
3 tables that we looked at yesterday, are you aware of
4 any similar report by the World Health Organization or
5 a like international body regarding any of those
6 pesticides apart from 2,4-D?

7 A. There have been registration
8 standards documents issued of various pesticides by the
9 United States Environmental Protection Agency over the
10 years, they are not an international body.

11 Q. I meant something like the World
12 Health Organization?

13 A. No.

14 Q. A similar in-depth look at the
15 scientific literature?

16 A. Not that I'm aware of.

17 Q. All right. Dr. Ritter, as we sit
18 here today in August, 1989, given the published views
19 of the scientific community on the public health risks
20 of 2,4-D and glyphosate specifically, can you indicate
21 to the Board whether it is your opinion that those
22 products can be used safely for forestry uses, if used
23 in accordance with the procedures authorized by the
24 federal registration system and label instructions?

25 A. There are a number of studies which

1 are ongoing particularly with reference to 2,4-D, both
2 in Canada and elsewhere. At the moment, certainly our
3 view that 2,4-D and glyphosate when used in accordance
4 with those label directions, should not constitute an
5 unacceptable degree of hazard, particularly in
6 the forestry sector.

7 Q. They are safe in the forestry sector
8 when used in accordance with authorized procedures?

9 A. Safe is a term which we tend not to
10 think of; we tend to think of varying degrees of risk,
11 and I prefer my answer to yours.

12 Q. Well --

13 A. We would not consider that that use
14 constitutes an unacceptable risk.

15 Q. All right. And that -- I understand
16 that answer from a representative of a federal
17 regulatory agency and I accept it as such, but what I'm
18 asking you, however, as a layperson in the context of
19 these hearings is:

20 Are members of the public entitled, in
21 your view and based on your knowledge in this area, to
22 assume, in light of the registration of those
23 pesticides, that those products are safe for use in the
24 forestry sector if used in accordance with properly
25 authorized procedures?

1 A. In the context in which you've asked
2 the question, I think the risks associated with the use
3 of these products would fall within a range that most
4 people would consider to be safe, yes.

5 Q. The answer was yes, Dr. Ritter?

6 A. Yes.

7 Q. Thank you. Is that also true of the
8 other pesticides authorized for use in the forestry
9 sector in Ontario based on your knowledge of them?

10 A. Based on my knowledge at this moment,
11 yes.

12 Q. All right. I will put it to you
13 quite simply: If there was any reliable or valid
14 information to the contrary, your agency would be
15 looking very hard at the question of -- or would in
16 fact have removed that registration; would they not?

17 A. That's correct.

18 Q. All right. These products are still
19 authorized for use out there and there is a reason for
20 that?

21 A. That's right.

22 Q. Thank you. And, finally, the second
23 report that you mentioned yesterday dealt with the
24 farmer motality study that I understand is underway?

25 A. That's right.

1 Q. And did I understand you correctly,
2 Dr. Ritter -- sorry, do I understand what you said
3 correctly that that is a study being conducted by
4 Health and Welfare?

5 A. That's correct.

6 Q. All right. And that -- just to recap
7 what you said about it yesterday, the only results to
8 date available are interim results emanating from the
9 investigations done in the Province of Saskatchewan?

10 A. That's correct.

11 Q. Thank you.

12 THE CHAIRMAN: Dr. Ritter, has there been
13 in your experience any situations where your agency has
14 approved for registration a product, felt obviously at
15 the time that the risk was reasonably low and,
16 therefore, could be used with no appreciable health
17 risk and then subsequently had to reverse your
18 position?

19 DR. RITTER: Yes.

20 THE CHAIRMAN: And I take it the reasons
21 for that were just additional studies which in effect
22 proved that your earlier assessment was wrong?

23 DR. RITTER: That's correct.

24 THE CHAIRMAN: And is that, to your
25 knowledge, the case with some of the spray programs

1 that I believe were conducted in eastern Canada?

2 DR. RITTER: No.

3 THE CHAIRMAN: There is one in Nova
4 Scotia I believe that recently was -- I think there was
5 a court case involving a particular spray program in
6 Nova Scotia whereby they obtained a court injunction in
7 terms of the use of that spray. Are you aware of that?

8 DR. RITTER: There have been a number of
9 legal actions particularly in the Maritimes in the last
10 10 or 12 years. I'm not specifically familiar with the
11 case that you cite, but...

12 THE CHAIRMAN: Okay, but those incidents
13 wouldn't be ones that would normally come to the
14 attention of your department?

15 DR. RITTER: They might. For example,
16 representatives of my unit were deeply involved in
17 what's become known as the 2,4-D trial, the injunction
18 hearing before the Nova Scotia Supreme Court some years
19 ago. We've been involved in other judicial proceedings
20 on a variety of matters.

21 THE CHAIRMAN: But it has never resulted
22 in your organization advocating the withdrawal of a
23 registration of 2,4-D?

24 DR. RITTER: No.

25 THE CHAIRMAN: Okay.

1 MR. MARTEL: Is there one not just going
2 on in the east coast within the last month in, I
3 believe, New Brunswick?

4 DR. RITTER: On 2,4-D?

5 MR. MARTEL: Yes.

6 DR. RITTER: Yes. I believe Mr. Jerry
7 White brought a motion in court seeking a permanent
8 injunction.

9 MS. MURPHY: In fairness to the witness
10 perhaps, and this is not necessarily something he would
11 normally know a lot of details about, and I am just a
12 little concerned that he is being asked to recount some
13 information that --

14 THE CHAIRMAN: Well, we are not
15 interested particularly, I don't think, in the court
16 action per se. I think the information we are trying
17 to obtain is whether his department, as a result of
18 these court actions, have concerns with 2,4-D -- with
19 the use of 2,4-D and the applications for which these
20 court actions concern.

21 DR. RITTER: We certainly have an
22 interest in the latter part of your question, and where
23 health effects become a central issue in a court
24 challenge we have always been involved because clearly
25 that's our responsibility. But many of these court

1 challenges that have taken place over the years, as you
2 can imagine, go outside of the realm of health effects.
3 They may deal with trespass, all kinds of law.

4 THE CHAIRMAN: No, I think we are just
5 interested really in the health effects and whether
6 your department in its involvement, if any, with any of
7 these court actions have had second thoughts or
8 concerns relating to the continued registration of this
9 product.

10 DR. RITTER: Never as a result of a court
11 action. We have certainly moved to take products off
12 of the market as a result of additional studies which
13 may become available subsequent to the initial
14 registration.

15 The most notable one we have referred to
16 during the course of these proceedings Alachlor, but
17 never as a result of a court action.

18 THE CHAIRMAN: Okay.

19 MS. CRONK: Mr. Chairman, the Nova Scotia
20 case to which you refer has come up a number of times
21 in the hearing. It is the Palmer decision, a decision
22 of Mr. Justice Nunn. It is our position that the Board
23 is entitled to take judicial notice of any reported
24 case anywhere in the jurisdiction of Canada and we
25 would propose to make a copy and provide them to the

1 Board so that you have it.

2 THE CHAIRMAN: Very well. Thank you, Ms.
3 Cronk. We were just basically trying to tie in the
4 results of those sort of notorious cases with any
5 action that your department might have taken or been
6 concerned with concerning the continued registration of
7 the products that are before this Board.

8 DR. RITTER: We took no action as a
9 result of that decision by Mr. Nunn in Nova Scotia.

10 MS. CRONK: Q. And I take it it follows
11 from that, Dr. Ritter, that notwithstanding any other
12 litigation in this country of which your department is
13 aware dealing with public health effects of 2,4-D, that
14 has not caused your department to seek, to rescind,
15 remove or tract or narrow the current registration
16 authorizations for the use of 2,4-D?

17 DR. RITTER: A. That's correct.

18 Q. That is also true with respect to
19 glyphosate; is it not?

20 A. Yes, it is.

21 Q. And that is also true with respect to
22 the other pesticides currently authorized for use in
23 forestry in Ontario?

24 A. That's correct.

25 Q. Thank you.

1 THE CHAIRMAN: Thank you.

2 MS. CRONK: My colleague Mr. Cassidy has
3 just reminded me - and I know, Mr. Chairman, that the
4 Board is aware of this - that the injunction request
5 was denied in the Palmer case, if that needed -- was of
6 assistance to have that drawn to your attention again.

7 THE CHAIRMAN: Thank you.

8 MS. CRONK: Q. Dr. Ritter, then,
9 referring to the farmer mortality - referring -
10 returning to the farmer mortality study that we were
11 discussing a moment ago, I am going to show you a
12 photocopy of an abstract form which was provided to me
13 by you yesterday and I would ask you whether this is
14 the information available on the public record in
15 official form concerning the results to date of that
16 study?

17 DR. RITTER: A. That is the document
18 that I provided to you yesterday including the
19 handwritten notation which I have added to the bottom.

20 Q. That is your handwriting at the
21 bottom?

22 A. That's my handwriting.

23 MS. CRONK: (handed)

24 THE CHAIRMAN: That will be Exhibit 717.

25 Thank you.

1 ---EXHIBIT NO. 717: Photocopy of abstract re:
2 farmer mortality study provided by
 Dr. Ritter.

3 MS. CRONK: Q. Dr. Ritter, before we
4 proceed to discuss the contents of this abstract, as
5 the photocopying of your handwritten notation was
6 faint, I wonder if you could simply read into the
7 record what your handwritten addition to the bottom of
8 the abstract is?

9 DR. RITTER: A. Yes. It says:
10 "A similar risk relationship was
11 established between expenditure on fuel
12 and risk of NHL independent of total
13 acres sprayed."

14 Q. Could I have it again, please?

15 A. "A similar risk relationship was
16 established between expenditure on fuel
17 and risk of NHL independent of total
18 acres sprayed."

19 Q. I will come back to that in a moment.
20 Am I correct, Dr. Ritter, that the first paragraph of
21 this abstract relates or sets out the background to the
22 study?

23 A. That's correct.

24 Q. And it indicates, and I quote:

25 "Public concern over the health hazards

1 of pesticides remains high with a major
2 focus on the risk of cancer in a
3 significant but lower level of concern
4 about adverse effects on pregnancy and
5 children."

6 Then the last sentence reads:

7 "This concern persists despite the
8 fact that there is little evidence of
9 physical health effects due to exposure
10 of the public to pesticides and other
11 environmental pollutants."

12 Stopping there for a moment, Dr. Ritter.

13 Who was the author of this abstract?

14 A. There were five of us. These are the
15 joint collaborators on this study which are drawn from
16 the Centre for Disease Control, Laboratory Centre for
17 Disease Control of Department of Health and Welfare and
18 the Environmental Health Directorate, my group of the
19 Department of Health and Welfare.

20 Q. All right. Were you then either one
21 of the five authors of this abstract or did you
22 supervise those persons from your department who were
23 involved in its production?

24 A. Both.

25 Q. All right. Do you agree with the

1 last sentence that I just read to you?

2 A. Yes, I do.

3 Q. All right. And then looking at
4 paragraph 2, as I understand it, the first sentence of
5 that paragraph sets out the reason for the study, that
6 being to assess the risks of cancer in relation to
7 pesticide exposure indices among the largest population
8 sub-group with direct, long-term occupational exposure;
9 that is to say, to assess the risk of cancer among
10 farmers?

11 A. That's correct.

12 Q. All right. And that for that purpose
13 the methodology adopted was to examine mortality among
14 365,000 farm operators identified in the 1971 census of
15 agriculture?

16 A. Yes.

17 Q. And having done that, to link it to
18 the national mortality database for the years 1971 to
19 1988 inclusive; that is to say, for a 16-year period?

20 A. Yes.

21 Q. And then the next sentence, as I
22 understand it, sets out the results in the Province of
23 Saskatchewan based on the study that has been carried
24 out, and am I correct that the results for the
25 Saskatchewan component indicate that farmers had lower

1 risks of death for all conditions combined and for
2 several major disease categories including all cancers
3 and including none-Hodgkins lymphoma than was expected?

4 A. That's correct.

5 Q. All right. Do I take from that then
6 that these results - and the numbers are actually here,
7 I have just read the text of the conclusion - that this
8 study in the Province of Saskatchewan demonstrates a
9 lower risk of mortality than would have been expected
10 in the grouping of farmers exposed to pesticides?

11 A. Yes.

12 Q. All right. And from the perspective
13 then of public health, human health risk, I take it
14 these test results are very encouraging; you'd agree?

15 A. Yes.

16 Q. And then, finally, in the next
17 sentence -- I'm sorry, and could I go back and add one
18 other thing. To the extent then -- to the extent then
19 that any press reports suggested contrary findings in
20 the Province of Saskatchewan as a result of this study,
21 those reports were inaccurate?

22 A. That's correct.

23 Q. These results in fact show the
24 lowering of risks of death for all conditions combined
25 and for all cancers?

1 A. That's correct.

2 Q. All right. You then go on to
3 indicate in the next sentence:

4 "However, on farms less than 1,000 acres
5 there was a statistically significant
6 exposure risk relation between risk of
7 non-Hodgkin's lymphoma and acreage
8 sprayed for weeds as reported in the 1971
9 census of agriculture."

10 Now stopping there. What does that mean?

11 In layman's terms, Dr. Ritter, what's being said there?

12 A. There are two things that are being
13 said in this paragraph. The first is that the acronym
14 in parentheses, SMR, is the standardized mortality
15 ratio which is the expression of the observed number of
16 deaths when compared to the expected number of deaths
17 over that period of time.

18 So that an SMR of less than one is an
19 expression of a desirable effect, if you like. An SMR
20 of less than one implies that fewer people died than
21 you would have predicted from national mortality
22 figures.

23 These SMRs that you see for deaths, for
24 all forms of cancer and for NHL specifically are all
25 less than one.

1 Q. NHL being non-Hodgkin's lymphoma?

2 A. That's correct. So for these three
3 variables in all three cases the actual number of
4 deaths observed was considerably below what one might
5 have expected based on averages for the Province of
6 Saskatchewan.

7 Q. And from a statistical perspective
8 that is an affirmative, a positive, an encouraging
9 result?

10 A. That's correct. To put it into
11 perspective, as I recall one might have expected about
12 16,000 deaths.

13 There were 70,000 farmers that were
14 examined in the Saskatchewan cohort. Of the 70,000
15 farmers for that period of investigation, from 1971 to
16 1988, one might have expected approximately 16,000
17 deaths in that cohort of 70,000 people. Of that
18 expectation of 16,000 we noted only approximately
19 12,000 deaths.

20 Q. All right, i.e., less than was
21 expected?

22 A. About one quarter less.

23 Q. All right.

24 THE CHAIRMAN: Is that from all causes?

25 DR. RITTER: That's death.

1 THE CHAIRMAN: Death.

2 MS. CRONK: Q. Well, death from what is
3 the Chairman's question.

4 DR. RITTER: A. Well, no, because we
5 then go on to talk about death from all forms of
6 cancer. But the first thing one does in this kind of a
7 study, because death is relatively easy to interpret in
8 terms of this kind of an investigation, we look at the
9 absolute possibility and that's just death.

10 Q. What was the standardized mortality
11 ratio for smaller farms, or do you remember?

12 A. I don't remember. The point really
13 that I was going to make was that the expected
14 incidence of non-Hodgkin's lymphoma -- the actual
15 incidence of non-Hodgkin's lymphoma in the Province of
16 Saskatchewan was somewhat less than the expected
17 incidence of non-Hodgkin's lymphoma.

18 I would not attach a great deal of
19 importance to the fact that it's slightly below one. I
20 would, for all practical purposes, say that the
21 incidence of non-Hodgkin's lymphoma among Saskatchewan
22 farmers was more or less comparable to what one might
23 have expected. It was no greater than what one might
24 have expected.

25 We found that observation noteworthy

1 because there are more phenoxy herbicides used in the
2 Province of Saskatchewan. I believe our latest
3 estimates suggest that there are more used in
4 Saskatchewan than in the rest of Canada combined.

5 Q. I wanted to ask you about that.
6 That, given the number of farmers in the province and
7 the extent of farming operations in that province; that
8 is, Saskatchewan, I take it you are saying that overall
9 there are more pesticides of that kind used in that
10 province than anywhere else in Canada?

11 A. I believe more are used in that
12 province than the rest of Canada combined.

13 Q. All right. That being the case and
14 these results being a reflection of the mortality
15 statistics for Saskatchewan, would you agree with me
16 that the results when available for other provinces in
17 Canada necessarily will be lower, given that the usage
18 is lower?

19 A. They will be --

20 Q. You would expect them to be?

21 A. They will be very difficult to
22 explain if they are not.

23 Q. All right. It would be your
24 expectation--

25 A. Yes.

1 Q. --that they would have to be lower?

2 A. Yes.

3 Q. All right.

4 A. Going on, once we've established that
5 the risk of non-Hodgkin's lymphoma in the Province of
6 Saskatchewan was actually comparable among farmers as
7 it was among non-farmers, we then went on to look at
8 what was actually driving that risk, however low it may
9 have been, and we made a couple of observations with
10 regards to what may have been driving that risk in
11 Saskatchewan.

12 Among other things, we noted that there
13 appeared to be a positive association between the
14 number of acres sprayed for weeds and the risk of that
15 non-Hodgkin's lymphoma even though the risk was no
16 greater than it was for people who don't farm at all.

17 Now, I emphasize that the relationship is
18 to a function we call spraying for weeds and it is not a
19 study of the relationship between the exposure to a
20 chemical and the risk of non-Hodgkin's lymphoma.

21 What I am trying to say is that this is
22 not a study of 2,4-D or glyphosate or anything else
23 specifically, it is a study of a practice, a function
24 on occupational activity as a function of risk and, in
25 this particular case, the function is spraying for

1 weeds.

2 But there are many variables in the
3 function that we call spraying for weeds, one of which
4 most certainly includes exposure to herbicides, but
5 it's only one of several variables. For example, one
6 would expect that there would be concomitant exposure
7 to a wide variety of solvents in that function we call
8 spraying for weeds, and so on and so forth.

9 It would, in my view, be premature and
10 incorrect to read that sentence to mean anything other
11 than what I just said. It is not a study of the
12 relationship between any specific chemical and any
13 specific disease.

14 We also found that there was a similar
15 positive risk relationship but not defined by any
16 acreage when one compared the dollars expended on fuel
17 oil which presumably we use as a surrogate for exposure
18 to fuel and the risk of non-Hodgkin's lymphoma.

19 Now, we felt what made that second
20 observation even more interesting than the first was
21 that in the first case, when we looked at acres sprayed
22 for weeds, the risk relationship fell apart beyond a
23 thousand acres and we don't have a ready explanation
24 for that.

25 In the first instance it's tempting to

1 speculate that that may be the case because on farms
2 larger than a thousand acres one might imagine that the
3 spraying operation would be contracted out and,
4 consequently, the individual farmer would no longer be
5 taking the risk and so the risk relationship might fall
6 apart on farms larger than a thousand acres. On
7 reflection we find that that may not be correct, it may
8 actually be the converse.

9 The information which we are gathering
10 suggests, at least in its preliminary phase, that it
11 may well be the smaller farms in which the spray
12 operation is contracted out because farmers with only a
13 few hundred acres may not be prepared to make the
14 investment in the spray rig necessary to spray their
15 own fields, and the larger farms may be spraying their
16 own fields.

17 So if that's true, I would have no
18 biologically plausible explanation for that observation
19 in spite of the fact that the observation stands.

20 Q. All right. Can I just see if I
21 understand all of this, Dr. Ritter, with respect to
22 this issue or at least what is important that be
23 understood from it. The first, as I understand it,
24 what you have said is that the relationship was found
25 to exist?

1 A. Yes.

2 Q. All right. It was found to exist
3 with respect to farms no larger than a thousand areas?

4 A. That's correct.

5 Q. All right. But do I also understand
6 you to have said, given the number of exposures or
7 components involved in the spraying of weeds, that one
8 should not conclude and it would be inaccurate to
9 conclude that the relationship exists because of
10 exposure to pesticides?

11 A. That's correct.

12 Q. Thank you. Can we leave it there, or
13 is there something more important that I should know
14 about that?

15 A. No, you can leave it there.

16 THE CHAIRMAN: Dr. Ritter, the other
17 statement you made about the expenditure of dollars on
18 fuel, are you making the assumption that the fuel
19 exposure is what, because the fuel would be used in
20 combines and tractors--

21 DR. RITTER: That's correct.

22 THE CHAIRMAN: --and there would be
23 long -- or exposure over long periods of time?

24 DR. RITTER: That's correct. The other
25 reason we were interested in the fuel relationship

1 specifically, now that you have asked the question, is
2 because there is experimental evidence in the
3 literature which suggests a possible association
4 between lymphoid tumors and exposure to these diesel
5 fuel components.

6 Lymphoid tumors were the ones which had
7 been implicated in some of the 2,4-D data in the last
8 five to ten years. So that there is substantial
9 biological plausibility, if you like, to the
10 observation of a risk relationship between what we're
11 measuring, which is expenditure which we are using as a
12 surrogate for exposure, and the risk of non-Hodgkin's
13 lymphoma. What strengthens that observation, in our
14 view, is the fact that it's independent of the number
15 of acres. So the more you buy the higher your risk,
16 plain and simple.

17 The earlier observation with regards to
18 the number of acres sprayed is less clear because the
19 relationship does fall apart at a specified acreage,
20 and unless and until we can come up with a rational and
21 biological explanation for why that should be so, it is
22 difficult in our view to attach a great deal of
23 importance to the observation in itself.

24 THE CHAIRMAN: Thank you.

25 MR. MARTEL: Were you able to track down

1 or did you attempt to trace the number of farmers who
2 sold their property and moved to, let's say, Victoria
3 or B.C. in attempting to get all of the deaths that
4 occurred, or you simply relied on Saskatchewan
5 statistics?

6 DR. RITTER: No, actually we didn't rely
7 on Saskatchewan statistics at all. The registry is
8 based -- the national mortality database, as the name
9 implies, is based on national mortality statistics. So
10 that if you die in Canada, regardless of where you
11 farmed, you show up in the national mortality database.

12 In addition, because this is a linkage
13 study, that vital statistic was then cross-linked to
14 your place of residence and occupation at the time that
15 you were living.

16 I might also add that we have, through a
17 cooperative arrangement, exchange information between a
18 number of U.S. states which are known to be popular for
19 Canadians to retire and they include, I believe, the
20 State of Florida, the State of Arizona, the State of
21 California, the State of New York, and I think there
22 are three others.

23 So that even if as a Canadian you were to
24 die in one of those states, that death statistic would
25 show up in our national mortality database.

1 MR. MARTEL: Thank you.

2 THE CHAIRMAN: Thank you. Ms. Cronk, are
3 you going to be much longer? How long do you think you
4 will be?

5 MS. CRONK: I will be I anticipate about
6 another ten minutes with Dr. Ritter and then I have
7 some questions for Mr. Kingsbury. Would you prefer to
8 rise now?

9 THE CHAIRMAN: I think we will take the
10 morning break at this time.

11 MS. CRONK: That's fine. Thank you, sir.

12 THE CHAIRMAN: 20 minutes. Thank you.

13 ---Recess taken at 10:00 a.m.

14 ---On resuming at 10:30 a.m.

15 THE CHAIRMAN: Thank you. Be seated,
16 please.

17 MS. CRONK: Thank you, Mr. Chairman.
18 Could we reserve a number, sir, for the extracts from
19 the World Health Organization report that I will have
20 photocopied and provide next week.

21 THE CHAIRMAN: Exhibit 718.

22 MS. CRONK: Thank you.

23 ---EXHIBIT NO. 718: Extracts from World Health
24 (Reserved) Organization Report.

25 MS. CRONK: Q. It also occurs to me,

1 really only as a suggestion to Ms. Murphy. That there
2 were some extracts from one of Mr. Kingsbury's paper
3 yesterday that were not -- the overheads were not
4 identical to those contained in the paper, and I recall
5 that she said she was going to have photocopies made
6 and perhaps she would like a number reserved.

7 MS. MURPHY: That would be wise. There
8 are a couple that are slightly different than the
9 slides, so it would just be photocopies of two or three
10 slides. One exhibit number would probably be
11 sufficient.

12 THE CHAIRMAN: Okay. Exhibit 719 for
13 that.

14 ---EXHIBIT NO. 719: Extracts from paper by
15 (Reserved) Mr. Kingsbury.

16 MS. CRONK: Thank you.

17 Q. Dr. Ritter, you will recall yesterday
18 during our discussion of the MOE expert panel report on
19 the carcinogenicity of 2,4-D I asked you whether the
20 report had been accepted by Ontario's Minister of the
21 Environment, Mr. Bradley, and your indication to the
22 Board was, yes, that was your understanding.

23 I have now obtained a copy of a news
24 release dated April 16, 1987 released by the Ontario
25 Ministry of the Environment with respect to that

1 report.

2 MS. CRONK: I would like that, sir, to be
3 the next exhibit.

4 THE CHAIRMAN: Exhibit 720.

5 ---EXHIBIT NO. 720: News Release from Ontario Ministry
6 of the Environment dated April 16,
1987.

7 THE CHAIRMAN: Dr. Ritter, while these
8 are being handed out, in this expert panel report,
9 Exhibit 714, there is a reference at the back under the
10 bibliography to the - just a moment - to the World
11 Health Organization 1984. It's on page 61. Is that
12 the report--

13 DR. RITTER: Yes, it is.

14 THE CHAIRMAN: --that you just dealt
15 with?

16 DR. RITTER: Yes.

17 THE CHAIRMAN: Thank you.

18 MS. CRONK: Q. Dr. Ritter, with respect
19 to this press release, as I understand it, if I could
20 attempt to summarize it and I would ask for your
21 confirmation of it, with the emergence in 1986 in the
22 United States of a number of studies, two in fact,
23 suggesting that 2,4-D might be a carcinogen, the
24 Ontario Minister of the Environment placed a moratorium
25 on the use in Ontario of new products containing 2,4-D

1 until a review of those studies could be conducted; am
2 I correct in that?

3 DR. RITTER: A. That's correct.

4 Q. And it was for the purposes of
5 reviewing that data and the existing state of the
6 scientific literature that the Minister appointed the
7 panel of experts who ultimately produced the report
8 that has been marked before this Board; is that
9 correct?

10 A. I believe that's correct, yes.

11 Q. All right. And this press release on
12 page 2 confirms; does it not, that that panel of
13 experts appointed by the Minister reviewed all current
14 studies including the two at issue from the United
15 States, being a report of the U.S. National Cancer
16 Institute and a report -- an epidemiological study from
17 the State of Kansas?

18 A. Those are the same studies.

19 Q. All right.

20 A. The study done in Kansas was
21 conducted by the U.S. National Cancer Institute.

22 Q. Sorry. And the second study then
23 that had sparked the issue, if I could put it that way,
24 in 1986 was a study completed by an industry task force
25 and that report as well was reviewed by the panel of

1 experts appointed by the Minister of the Environment?

2 A. That's correct.

3 Q. All right. And am I also correct
4 that in this press release the Environment Minister,
5 Jim Bradley, announced that new products containing the
6 herbicide 2,4-D would be allowed in Ontario as and from
7 that date?

8 A. Yes.

9 Q. And the press release goes on to
10 indicate that this decision followed from a
11 recommendation made to the Minister by the Ontario
12 Pesticide Advisory Committee; is that correct?

13 A. Yes.

14 Q. And that committee had reviewed the
15 expert panel report on 2,4-D carcinogenicity which has
16 been marked as an exhibit before this Board?

17 A. Yes.

18 Q. And the press release indicates that
19 a Dr. John Doole of the Department of Pharmacologyy,
20 Toxicology and Therapeutics at the University of Kansas
21 reviewed the expert panel report on behalf of the
22 Ontario Pesticide Advisory Committee. Am I right?

23 A. Yes.

24 Q. And that based on the conclusions of
25 the expert panel, the advisory committee advised the

1 Minister:

2 "No changes be made to current provincial
3 regulatory requirements governing the use
4 of 2,4-D at this time and, further,
5 OPAC also advised the Minister to lift
6 the ban on new 2,4-D products."

7 A. That's correct.

8 Q. Those were the recommendations of the
9 committee?

10 A. Yes.

11 Q. And they followed upon a review and
12 consideration of the expert panel report?

13 A. Yes.

14 Q. All right. Am I correct then that
15 this press release confirms that the Minister of the
16 Environment of the day accepted the report, received
17 the recommendations of his advisory committee in light
18 of the report and acted upon them accordingly?

19 A. Yes.

20 Q. Thank you. There is one further
21 report, Dr. Ritter, that I would ask for your comment
22 and assistance on. It deals with the issue of
23 insecticides.

24 It is an article published in January of
25 1984 in a publication entitled: Applied and

1 Environmental Microbiology. It is entitled:
2 Assessment of the Potential of Insecticides,
3 Emulsifiers and Solvent Mixtures to Enhance Viral
4 Infection in Cultured Mammalian cells.

5 And I understand that you were one of the
6 study coordinators for that study that resulted in the
7 ultimate publication of the article. Is that correct?

8 A. That's correct.

9 MS. CRONK: (handed)

10 THE CHAIRMAN: Exhibit 721.

11

12 ---EXHIBIT NO. 721: Article entitled: Assessment of
13 the Potential of Insecticides,
14 Emulsifiers and Solvent Mixtures
15 to Enhance Viral Infection in
Cultured Mammalian Cells
published in Applied and
Environmental Microbiology.

16 MS. CRONK: As I understand it, Dr.
17 Ritter, there were two study coordinators for the
18 purpose of this study. You were one, the other was Dr.
19 D. G. Ecobichon; is that correct?

20 DR. RITTER: A. That's correct.

21 Q. Do you see Dr. Ecobichon in the room
22 today?

23 A. He's one over from you.

24 Q. Thank you. And Dr. Ecobichon; am I
25 correct, is attached to the Department of Pharmacology,

1 Toxicology and Therapeutics at McGill University in
2 Montreal?

3 A. That's correct.

4 Q. All right. Can you explain to the
5 Board, please, what the purpose of this study was?

6 A. In the early 1980s, latter 1970s,
7 there had been a number of reports originating at
8 Dalhousie University in Halifax which suggested that
9 there may be an association between the use of
10 insecticides in the Maritime province -- in the
11 Maritime Provinces in the forestry spray programs and
12 the incidence of Reye's Syndrome which is a pediatric
13 disorder frequently resulting in death in those
14 provinces.

15 We became interested in it. Dr.
16 Ecobichon and I became interested in that possibility
17 and thought that it might be useful if we could be, in
18 the first instance, attempt to replicate some of the
19 results which had been -- some of the experiments which
20 had been published by these investigators from
21 Dalhousie University and, in the second place, to
22 perhaps advance that technology somewhat.

23 We undertook that study, therefore, and
24 to try to ensure the most reliable results we had the
25 study protocol varified by the initial study author,

1 that was Dr. Kenneth Rozee who at the time was with the
2 Department of Microbiology at Dalhousie.

3 Upon verification of our protocol by Dr.
4 Rozee, we then approached three independent
5 laboratories which included Bio Research Laboratories
6 in Montreal, the largest contract research facility in
7 Canada.

8 Dr. C. Y. Chang, who was head of the
9 Division of Microbiology and Immunology at the Ottawa
10 General Hospital and Dr. J. Thorson who was with the
11 Division of, I think, Infectious Disease at the Ontario
12 Veterinary College.

13 Q. Why three laboratories?

14 A. Simply to increase the precision, the
15 reliability of the result. I mean, one could have had
16 it done in one, but we operated on the assumption that
17 if we did it in three the results would be more
18 reliable than in one.

19 Q. At whose behalf or whose request was
20 the study carried out?

21 A. The study was really done at our
22 initiation but was funded by Forest Protection Limited
23 in New Brunswick.

24 Q. All right. And did the Canadian
25 Forestry Service have any involvement or interest in

1 the work?

2 A. These are events that took place now
3 almost seven years ago. They had interest in it, I
4 don't know that I would say they had involvement in it.

5 Q. All right. Perhaps, in fairness to
6 you, could I refer you to the first paragraph of the
7 article, approximately 10 -- 15 lines down in which it
8 indicates:

9 "In an attempt to replicate results
10 previously published..."

11 Stopping there. I take it that the
12 previously published results were those which were used
13 in an effort to associate insecticide spraying in New
14 Brunswick with an incidence of Reye's Syndrome?

15 A. That's correct.

16 Q. All right.

17 A. And if you were to check citation No.
18 5 it refers to K. R. Rozee the investigator that I
19 referred to few moments ago.

20 Q. So the reports were his?

21 A. That's right.

22 Q. All right.

23 THE CHAIRMAN: Dr. Ritter, is this the
24 same disease associated with -- sometimes with
25 teenagers taking aspirin?

1 DR. RITTER: Yes, it is. It was
2 subsequent to this work, I might even be so bold to
3 suggest that in part because of this work, that the
4 U.S. Centre for Disease Control and the U.S. National
5 Institutes of Health undertook some rather ambitious
6 studies to look at the incidence and possible etiology
7 of Reye's Syndrome in the United States and after a
8 rather extended period of investigation, came to the
9 conclusion that the primary factors involved in the
10 etiology of Reye's Syndrome were aspirin following a
11 particular illness.

12 And subsequent to that, as you know, both
13 the American and Canadian Pediatric Academies have
14 recommended that children at risk do not take aspirin
15 with these diseases.

16 THE CHAIRMAN: Thank you.

17 MS. CRONK: Q. Continuing on in the
18 sentence to which I have directed your attention, it
19 indicates; does it not, that the Canadian Forestry
20 Service had suppressed an interest in having the
21 experiment outlined in the previously published
22 literature validated?

23 DR. RITTER: A. That's correct.

24 Q. All right. And that, in part I take
25 it, led to your interest and that of Dr. Ecobichon in

1 carrying out this study and in devising the protocol
2 for the study?

3 A. That's correct.

4 Q. All right. And then you were
5 explaining to the Board that you approached and
6 arranged for three independent laboratories to carry
7 out the appropriate tests?

8 A. That's right.

9 Q. Am I correct that of the components
10 tested, they included three chemical insecticides
11 Madacil, 7, and Fenitrothion?

12 A. That's right.

13 Q. All right. And could you outline as
14 reflected in the discussion section of the paper,
15 beginning over at page 83, could you outline to the
16 Board the results of the study that you conducted with
17 Dr. Ecobichon?

18 A. Essentially we found two critical --
19 we made two critical observations from the study. I
20 should perhaps just add, in terms of the protocol,
21 again in our attempt to make the results as reliable as
22 possible, the study was done in a double blind fashion;
23 that is, neither Dr. Ecobichon nor I nor the
24 investigators had any knowledge of the chemicals that
25 they were testing. They were individually coded and

1 the codes for the three participating laboratories were
2 different.

3 So that Code A, for example, for Lab 1
4 contained a different chemical than Code A for Lab 2 so
5 that there could not be any collaboration between
6 laboratories or between the study coordinators; that
7 is, Dr. Ecobichon and myself, and the laboratories.
8 That code was only disclosed after the results were in.

9 Q. All right.

10 A. The results -- the experiments
11 essentially provided us with two sets of observations.
12 The first and perhaps foremost was that we were
13 ununable to replicate the results which Dr. Rozee had
14 published previously based on a protocol in which he
15 had participated in its development and had
16 subsequently approved.

17 Q. All right. Stopping there for a
18 moment. In the scientific community, as part of a
19 scientific investigation, what is the significance of
20 replication or lack there of results?

21 A. It's absolutely essential. In the
22 absence of an ability to replicate results, I think one
23 would have to wonder very seriously about the validity
24 of the result. It's the hallmark of scientific
25 investigation.

1 Q. And the second observation?

2 A. The second observation we made is
3 that none of these agents essentially had the capacity
4 to enhance viral replication and, what I mean by that
5 is, this protocol was designed to study the capacity of
6 these agents, the ability of these agents to allow
7 viruses to replicate at rates that would be considered
8 to be unusual; that is, to enhance viral infection,
9 that was the intent of the study.

10 The first observation, as I said, was
11 that we were unable to replicate the result. The
12 second observation was notwithstanding the first none
13 of the agents tests, and there was I believe some
14 combination of 40 odd agents that were tested in this
15 study, none of the chemicals tested had the capacity to
16 increase the potential for viral infection.

17 Q. Including the three chemical
18 insecticides to which I drew your attention a moment
19 ago?

20 A. Including the three chemicals.

21 Q. All right. Bearing in mind then the
22 two observations that flowed from this work, what is
23 your opinion regarding the suggestion reported in the
24 scientific literature in the early 1980s regarding a
25 possible association between insecticide spraying and

1 Reye's Syndrome?

2 A. In our view there is no association.

3 Q. Are you aware of any subsequent
4 scientific work or investigation which causes you to
5 reconsider that opinion in any way?

6 A. No.

7 Q. Are you aware of any such work which
8 cast doubt upon the results obtained in this study?

9 A. No.

10 Q. What is the area of Dr. Ecobichon's
11 expertise?

12 A. Pharmacology, toxicology.

13 Q. Is he an acknowledged expert, in your
14 view, in those fields?

15 A. Yes, he is.

16 Q. With particular expertise in
17 insecticides?

18 A. Yes.

19 Q. Thank you.

20 THE CHAIRMAN: Dr. Ritter, how would a
21 suspicion arise in the first place if it wasn't based
22 on some scientific study?

23 When you refer broadly to: There was
24 some indication in the scientific literature that there
25 was a correlation between Reye's Syndrome and some of

1 these agents, how would that conclusion be reached so
2 that it would reach scientific -- the status of being
3 considered part of the scientific literature in the
4 first place if there weren't studies that would tend to
5 show perhaps opposite results to what you found?

6 DR. RITTER: The impetus for the work
7 came originally in the latter 1970s when Dr. John
8 Crocker, who is a pediatric nephrologist at the
9 Children's Hospital in Halifax suggested that he felt
10 that there were an unusually large number of cases of
11 Reye's Syndrome in the Maritime Provinces.

12 The Children's Facility in Halifax being
13 the regional referral centre for pediatric medicine
14 would have put Dr. Crocker in the unique position of
15 having examined virtually all of the children with the
16 disease in the Maritime Provinces.

17 They felt, at least on their preliminary
18 observation, that there appeared to be a higher
19 incidence than they may have expected. That led them
20 to a series of experimental trials on which they based
21 their original hypothesis that there was an association
22 between the two events.

23 In our view the initial observation from
24 the cases admitted at the hospital as well as the
25 subsequent experimental trials were flawed both in

1 their logic and in their conduct and, consequently, we
2 were prompted to what we felt was to undertake a
3 somewhat more exhaustive and more credibly scientific
4 approach to that investigation.

5 For example, in the case of the human
6 trials -- we felt that in the case of the human
7 disease, we felt that the number of cases of Reye's
8 Syndrome that might occur in the Maritime Provinces was
9 probably too low, below the threshold, because the
10 disease has a relatively -- is relatively uncommon, has
11 a very low background rate and the population at risk
12 in the Maritime Provinces we felt was sufficiently
13 small and that it was unlikely that even if the disease
14 were going on, it would necessarily be expressed. So
15 we felt that this kind of thing really required a more
16 intensive investigation.

17 I should add perhaps that, which has not
18 become evident here, we were not the only ones that
19 were asked to examine this issue. Dr. Walter Spitzer
20 who was Chief of Medicine at McGill University at the
21 time was also asked by the Government of New Brunswick
22 to examine the possible association between Reye's
23 Syndrome and the forestry spray programs in the
24 Province of New Brunswick.

25 And to summarize just from memory,

1 essentially what Dr. Spitzer concluded at the time
2 after his investigation was that he could find no
3 evidence of any possible association between the two
4 events.

5 THE CHAIRMAN: Would it be normal in
6 scientific circles after you completed a study such as
7 the one you coordinated to either correspond or consult
8 with the authors of a previous study that came to an
9 opposite conclusion? Would there be discussion of,
10 say, your results with the results obtained by those
11 preceding you which came to opposite conclusions? For
12 instance, have you talked to this doctor in Halifax--

13 DR. RITTER: Yes.

14 THE CHAIRMAN: --since your study, in the
15 light of your own results?

16 DR. RITTER: Yes. There were discussions
17 which ensued between ourselves and Dr. Rozee, both
18 during the conduct of the study as well as in the
19 period immediately after the study, as well as during
20 the time that Dr. Spitzer was investigating the issue
21 as well.

22 THE CHAIRMAN: And what would your
23 opinion be as to the state of the scientific opinion on
24 this issue at this time as a result of the initial
25 study by this doctor in Halifax plus the two subsequent

1 studies?

2 DR. RITTER: Well, I should perhaps
3 remind you that we had approached Dr. Rozee and asked
4 him to participate in the conduct of this study. We
5 had in fact requested that he be laboratory No. 4.

6 He indicated at the time that he was busy
7 and that the undertaking would be expensive. We
8 offered at the time to underwrite any costs, including
9 the provision of additional staff so that it would not
10 create a burden on his workload to participate in the
11 study.

12 We felt it would have been very important
13 to have Dr. Rozee as one of the four collaborators in a
14 double blind study of this type. He declined that
15 offer.

16 I think it would be fair to say that
17 following our investigation and that of Dr. Spitzer's,
18 and not necessarily because of it, there has been very
19 little in the literature, if anything, other than the
20 odd isolated report from the initial laboratory of Dr.
21 Rozee's in which there has been any further attempt to
22 associate Reye's Syndrome and forestry insecticide
23 spray programs.

24 THE CHAIRMAN: Thank you.

25 MRS. KOVEN: Was Dr. Spitzer's work

1 epidemiological or did it involve some lab tests and
2 diagnostic work?

3 DR. RITTER: It involved considerable
4 diagnostic work. Dr. Spitzer is a clinician and, at
5 the risk of using a cliché, I'm glad you asked that
6 question.

7 One of the initial observations that Dr.
8 Spitzer made -- he had requested charts of patients and
9 there was quite a bit of controversy at the time
10 because he was initially denied access to those charts
11 from the Children's Hospital. He felt very strongly
12 that he needed to see those charts and felt that the
13 denial to access those charts was really a violation of
14 a trust because he too was a physician and that there
15 was really no legitimate reason why he should be denied
16 access to those charts.

17 He went on to conclude in his report to
18 the Government of New Brunswick in answer perhaps, and
19 further answer to the Chairman's question, that part of
20 the reason why these investigators may have arrived at
21 an erroneous conclusion is because the data which they
22 used had been seriously flawed.

23 He found, for example, that of the
24 substantial number of cases of Reye's Syndrome which
25 they had examined at the Children Hospital in Halifax

1 were in fact not from the region that had been sprayed
2 at all, in fact a number weren't even Canadian.

3 Now, if one makes the assumption that
4 your cases are all exposed and one establishes a risk
5 relationship on that basis, obviously you are going to
6 arrive at a very different conclusion than if a
7 substantial proportion of those cases had never been
8 exposed at all. And, in fact, that is exactly what had
9 happened in the case of the Rozee investigation. There
10 were a significant number of his cases that had never
11 been exposed to these forestry insecticide spray
12 programs at all.

13 That was an omission which Dr. Spitzer
14 felt was rather serious -- an error which he felt was
15 rather serious and, among other things, had requested
16 that there be a correction issued through the Canadian
17 Medical Association Journal and although the correction
18 ultimately was prepared by Dr. Rozee and submitted to
19 the Journal, needless to say it's the original
20 publication that stands and one rarely references the
21 correction.

22 So that the original paper suggests an
23 association between Reye's Syndrome in children and the
24 forest industry spray program and the fact that that
25 conclusion is probably entirely erroneous is not nearly

1 as evident as the original suggestion.

2 There were a number of other flaws in the
3 logic, as I say, that developed during the course of
4 Dr. Spitzer's investigation, but that was I think
5 potentially perhaps the most serious.

6 MRS. KOVEN: The problem with
7 establishing exposure isn't usually that
8 straightforward.

9 DR. RITTER: Well, what it was really
10 based on was -- their investigation was based on the
11 assumption that these children had come from areas that
12 had been regularly sprayed with forestry insecticides.

13 It became evident during the course of
14 Dr. Spitzer's work that many of these children had
15 never been near any area that had been sprayed with
16 forestry insecticide.

17 So that I think Dr. Spitzer attempted to
18 eliminate that element of doubt by looking at -- rather
19 than looking at probably exposed categories, he looked
20 at in all likelihood never exposed as compared to
21 possibly exposed. So that I think he gave the benefit
22 of the doubt to the investigation, if you like, but it
23 was evident that there were a significant number of
24 cases that it was virtually impossible to imagine how
25 they ever would have been exposed to any of these

1 sprays.

2 There was another rather serious
3 microbiological error in some of the assumptions which
4 Dr. Rozee and his investigators had made as well, and I
5 won't belabour you with what that was, unless you want
6 to hear it. But, again, a number of people involved in
7 infectious disease who had looked at that work,
8 including Dr. Chang, felt that the hypothesis was
9 sufficiently flawed in its foundation that the
10 conclusion was hardly credible.

11 MRS. KOVEN: And, therefore, there is no
12 perspective follow up on that data or on that
13 population of children?

14 DR. RITTER: It would be very difficult
15 to follow that population in any case because, as I
16 indicated, given the background incidence of Reye's
17 Syndrome and given the population at risk in the
18 Maritimes, it is unlikely that one would be able to
19 detect an increase unless it were virtually epidemic
20 in a population of that size.

21 But the Centre for Disease Control in the
22 United States in Atlanta did undertake an investigation
23 on the etiology of Reye's Syndrome and their conclusion
24 was that probably the single largest factor, if not the
25 sole factor, that was driving the incidence of Reye's

1 Syndrome at least in the United States was the use of
2 aspirin in children particularly who were affected
3 either by influenza-B or varicella chicken pox.

4 And, as you know, there have been
5 warnings that have been issued since that time that
6 have suggested that -- well, depends on academy you
7 follow. Some have suggested that aspirin not be used
8 in children at risk age at all because you really don't
9 know early on in the disease if they have chicken pox
10 or influenza-B. Other sources have been somewhat less
11 concerned and said that if there is a diagnosis of
12 either influenza-B or chicken pox, aspirin should not
13 be used as an analgesic in these children. They both
14 mean essentially the same thing, one is a little more
15 cautious than the other.

16 But, as I indicated, to the Chairman, I
17 know of no work, credible or otherwise, that has been
18 published since this time other than the odd isolated
19 report which followed immediately after this work which
20 has suggested an association between these kinds of
21 agent and Reye's Syndrome.

22 MRS. KOVEN: Was Spitzer able to
23 establish a profile of exposure in the paths that would
24 identify specific changes in the use of sprays or the
25 product composition? Often in that sort of study they

1 are able to say: Yes, there was a window of exposure
2 that changed significantly over time as practices
3 improved. So in fact, the concern would be very much a
4 past exposure.

5 DR. RITTER: But Reye's Syndrome is not a
6 disease of chronic onset and one would not have
7 expected that a historical alteration in exposure
8 potential would have altered the incidence of the
9 disease; that is, an exposure practice which may have
10 been in Vogue ten years ago, an exposure scenario that
11 may have been popular 10 years ago is unlikely to
12 affect the incidence of Reye's Syndrome today.

13 It is a disease which is caused by
14 whatever the agent is, and let's assume it's aspirin,
15 it's a disease which is caused by very, very
16 contemporary exposure to the agent unlike cancer. So
17 that the kind of suggestion you make would probably not
18 be very useful in this kind of a disease.

19 MS. CRONK: Q. Dr. Ritter, at the risk
20 briefly of inviting you to be modest, is it fair to say
21 that the work done by Dr. Ecobichon and yourself, as
22 reported in this paper, put to rest the suggestion in
23 the scientific community of an association between
24 Reye's Syndrome and use of these insecticides?

25 A. I think I would prefer to say that

1 the work that we coordinated, the work that we did
2 suggested that the original experimental protocol was
3 not credible. I don't know that we made a useful
4 contribution to the scientific literature other than to
5 establish the validity of an earlier protocol.

6 Q. Well perhaps others, apart from
7 yourself, will be prepared to draw some inference from
8 the fact that it hasn't been dealt with in the
9 literature since.

10 There is one other aspect, though, of the
11 work that Dr. Spitzer did in light of your conversation
12 with Mrs. Koven that I would like to pursue.

13 It is my understanding that Dr. Spitzer
14 had another set of experiments done similar to those
15 which Dr. Ecobichon and yourself had arranged to have
16 done in this report and that they were conducted at
17 laboratories in the midwest United States. Are you
18 familiar with those?

19 A. Yes, I am.

20 Q. And what were the results of that
21 duplication of your study experiments, if you will?

22 A. They were similar to our own.

23 Q. All right. I would like the purpose
24 for bringing this to the attention of the Board to be
25 clear in my discussion with you, Dr. Ritter.

1 Would it be fair to suggest that the
2 controversy initially occasioned by the published
3 literature suggesting an association between Reye's
4 Syndrome and the use of insecticides is an example of
5 the kind of issue that can arise from time to time in
6 the scientific community concerning the public health
7 aspects of a variety of chemical agents?

8 A. Yes.

9 Q. It happens from time to time?

10 A. It happens with some regularity and
11 frequency.

12 Q. I'm glad you said that. And is it
13 also not the case -- well, when an issue of that kind
14 arises, particularly with respect to the public health
15 implications of chemical agents, is it fair to say that
16 they become and receive critical public health
17 attention because there's the possibility that they may
18 be true?

19 A. Yes.

20 Q. All right. And that leads, does it
21 not, on occasion to concerns in the minds of the public
22 about the possible public health effects of the use of
23 chemicals that, in many instances, are not from a
24 scientific point of view valid?

25 A. That's correct. Our motive, as I

1 mentioned, for undertaking this work, for example, was
2 not to discredit Dr. Rozee or Dr. Crocker; our motive
3 was because they had presented a published hypothesis
4 that suggested a possible association between a very
5 serious and often fatal disease and the use of
6 insecticides, and that was a possibility that I felt we
7 had to deal with very seriously.

8 Q. Because, were it true, it would be a
9 very serious public health concern; isn't that so?

10 A. That's correct.

11 Q. All right. And isn't the very issue
12 that I'm putting to you now what your group was
13 alluding to in the introduction to the abstract from
14 the farmer mortality study when you indicate that a
15 concern over the health hazards of pesticides remains
16 high in the public, notwithstanding the fact that there
17 is little evidence of physical health effects due to
18 exposure of the public to pesticides and other
19 environmental pollutants?

20 A. That's correct. We see the role of a
21 public health agency to be -- to include investigation
22 of any plausible hypothesis of an association between
23 human disease and use practices, in my particular case,
24 in association with use of pesticides, and because the
25 possible risks associated with pesticide use are

1 plausible, these agents are, after all is said and
2 done, toxic agents often intended to kill something,
3 it's a plausible hypothesis.

4 And, consequently, we take those kinds of
5 suggestions, such as the ones made by Drs. Rozee and
6 Crocker and others that have made suggestions with
7 regard to farmers and pesticides, we take those
8 suggestions very seriously.

9 Q. And you would agree with me that that
10 clearly is appropriate because they could represent
11 real public health hazards--

12 A. Yes.

13 Q. --were they substantiated?

14 A. That's correct.

15 Q. Yes.

16 THE CHAIRMAN: Dr. Ritter, in fairness
17 though, on the other side of the coin, there are
18 examples like thalidomide and others which have been
19 studied, licensed, put on the market and subsequently
20 scientifically proved to be quite harmful.

21 DR. RITTER: That's correct.

22 THE CHAIRMAN: Would you not agree?

23 DR. RITTER: That's correct.

24 MS. CRONK: Q. And lest the import of
25 the questions that I have put be misinterpreted, I am

1 suggesting to you, Dr. Ritter, that that is the point
2 of the exercise of a public health agency, that it's
3 important that you respond to and investigate any
4 suggestions of that kind to determine whether in fact
5 they are validly and soundly based?

6 DR. RITTER: A. Yes, I would just add
7 the caveat, plausible suggestions.

8 Q. Yes. And this was a case where it
9 was plausible, it was investigated and, based on the
10 work that has been done, it has been found to be
11 unfounded or scientifically invalid; would you agree?

12 A. That's correct.

13 Q. Thank you. And then just one final
14 aspect of that dealing with fenitrothion, one of the
15 insecticides specifically tested in the study that you
16 did with Dr. Ecobichon.

17 I asked you yesterday in a totally
18 different context whether there were any applications
19 which had led to approval of a registered pesticide in
20 this country intended purely for forestry use
21 application, you recall that?

22 A. Yes.

23 Q. And I believe you responded to me
24 that fenitrothion was an example of one of those?

25 A. Yes.

1 Q. Am I -- in thinking about that
2 further last evening, Dr. Ritter, am I correct that at
3 the time that the registration for fenitrothion in
4 Canada was granted, it had a very extensive
5 agricultural use experience international?

6 A. That's correct.

7 Q. Across the world?

8 A. That's correct.

9 Q. And that there was a considerable
10 amount of data concerning both its health effects and
11 other aspects of its usage arising from that
12 agricultural usage experience?

13 A. Yes.

14 Q. There was an existing database?

15 A. Yes.

16 Q. And is it fair to say that that
17 existing database formed part of and was certainly
18 available for the purposes of the evaluation --
19 registration evaluation process here in Canada?

20 A. Yes.

21 Q. All right. It would, I suggest to
22 you, be inappropriate and erroneous - I don't suggest
23 that you suggested this - but it would be inappropriate
24 to conclude that all of the work which you have
25 described to the Board need be done to put together a

1 registration package in this jurisdiction was done for
2 fenitrothion simply for the purposes of a forestry use
3 application?

4 A. That's correct.

5 Q. Thank you. Dr. Ritter, those are my
6 questions of you and I thank you.

7 Mr. Kingsbury, I wonder if I could turn
8 to you now, sir, by way of a number of preliminary
9 matters, and then I have some questions for you
10 concerning the ESSA Document itself and certain of its
11 content.

12 If I could deal with the preliminary
13 matters first. You will recall, Mr. Kingsbury, that
14 you spent some time yesterday morning explaining to the
15 Board the environmental impact assessment strategy that
16 is applied to forestry pesticides. Do you recall that?

17 MR. KINGSBURY: A. Yes, I do.

18 Q. All right. And one of the exhibits
19 that you provided to the Board was Exhibit 711D which
20 is a summary of that strategy as applied to forestry
21 pesticides and you explained in detail what is involved
22 in each of the items identified in that exhibit?

23 A. That's correct.

24 Q. All right. You referred a number of
25 times as I took it down in my notes, Mr. Kingsbury -

1 and I perhaps didn't get it down accurately - but I
2 thought you referred a number of times to "we" in the
3 sense of: "we" used an indicator species approach, for
4 example, "we" picked a species that we knew we were
5 able to study upon which there was data available,
6 examples of that kind. Do you recall that?

7 A. That's correct.

8 Q. All right. What I was unclear about,
9 Mr. Kingsbury, and I would ask for your clarification:
10 When you described to the Board the strategy applied to
11 environmental impact assessments for pesticides --
12 forestry pesticides, as outlined on this exhibit, were
13 you talking about the strategy that was employed --
14 that is employed by the federal government, that is
15 employed by the Ministry of Natural Resources, that was
16 employed by the ESSA group, or is one that you propose?
17 Where does it fall in those possibilities?

18 A. I guess the most appropriate answer
19 to what I meant by the royal "we" was the strategy that
20 has been in place and has involved within the
21 federation registration process in Canada. It would be
22 a strategy that has come from the ongoing interaction
23 of the various agencies that I spelled out who play a
24 role in terms of assessing environmental risks.

25 Q. And was the work done by the group of

1 experts who produced the ESSA report consistent with
2 that approach to environmental impact assessment?

3 A. The ESSA -- the people who worked on
4 the ESSA Document basically reviewed a literature base
5 that reflected the fact that that is the strategy in
6 place in assessing environmental impacts of forestry
7 pesticides.

8 Q. All right. It was then consistent
9 with that approach?

10 A. Yes.

11 Q. Yesterday as well, a number of times
12 in the course of your description to the Board of the
13 result of the ESSA investigations concerning potential
14 impacts, you described what you said were observed
15 effects of chemical insecticides. Do you recall that
16 generally?

17 A. Yes.

18 Q. By way of an example, one of the
19 descriptions that I recall you dealing with was the
20 observed effects of chemical insecticides on aquatic
21 invertebrates in forest ponds and static waters and you
22 compared for the Board what the effect of aminocarb
23 was, carbaryl and fenitrothion in relationship to one
24 another. Do you recall that?

25 A. That's correct.

1 Q. All right. Would you agree with me,
2 Mr. Kingsbury, that the approved application rate for
3 chemical insecticides in Ontario varies from chemical
4 to chemical?

5 A. Yes, absolutely.

6 Q. So that when one attempts to make
7 toxicity comparisons as among the chemical insecticide
8 group it is important to recall and to understand that
9 the authorized application rates of those chemicals do
10 differ?

11 A. Absolutely.

12 Q. All right. And I guess what I am
13 suggesting to you, inferentially at least, and now
14 expressly, Mr. Kingsbury, is that in looking at the
15 comparative impact of chemical insecticides in terms of
16 their effect on any particular part of the environment
17 one should with great caution make blanket or
18 generalized toxicity comparisons because the chemicals
19 are different?

20 A. I agree.

21 Q. As are the application rates and the
22 circumstances of their use?

23 A. Yes.

24 Q. Thank you. Then yesterday --
25 following from your evidence yesterday as well there

1 was a discussion, you may recall, when you outlined for
2 the Board what was meant by bio-concentration and
3 bio-magnification as referred to and defined in the
4 ESSA Document. Do you recall that?

5 A. Yes, I do.

6 Q. All right. There was one element of
7 your evidence that I wished to have clarified. At one
8 point, as I took it down in my notes - and I indicate
9 immediately I may have not recorded it accurately - but
10 I understood a question to be put to you by the Board
11 to this effect: Are you saying that there isn't any,
12 meaning bio-concentration or bio-magnification, or that
13 you wouldn't register them if they did occur.

14 Do you remember the question, let me just
15 put it that way?

16 A. Yes, I recall the question.

17 Q. All right. There were two elements
18 to that question. The first is: Does it happen, and
19 the second is: If it did, would they be registered.

20 As I understood your testimony yesterday
21 you indicated, and I would ask for your confirmation,
22 that with respect to the pesticides in use in Ontario
23 there is no evidence that bio-concentration or
24 bio-magnification occurs; is that correct?

25 A. Could you just repeat that so I am

1 clear?

2 Q. Yes. I understood your evidence to
3 be yesterday that with respect to the pesticides used
4 in forestry in Ontario in forestry situations, there is
5 no evidence that bio-magnification or bio-concentration
6 occurs. Did I understand your evidence correctly?

7 A. I believe so, although I may have
8 dealt with those individually and I believe when I came
9 back and talked to the Board and said I had used the
10 royal "we" and talked about those, that I specified
11 there is no evidence of significant bio-concentration
12 and significant bio-magnification.

13 Q. All right. Well, that's the
14 refinement of that answer that I am interested in, Mr.
15 Kingsbury. You have added the word significant
16 bio-concentration or bio-magnification because, as I
17 understood it, you told the Chairman yesterday that if
18 they existed, those compounds, those chemicals would
19 not be registered?

20 A. That's very much the belief that -- I
21 said to the Board that if there is evidence of
22 significant bio-concentration or bio-magnification it
23 would, in fact, lead to a product not receiving
24 registration.

25 Q. Yes. And would you agree with me

1 that the important -- one of the important features of
2 what you just said is the word significant?

3 A. I would agree in that we are dealing
4 with a rather complex issue, and I didn't go into it at
5 length yesterday.

6 When I say that, the idea of measuring
7 bio-concentration in terms of the relationship as it is
8 defined, as we are using the word here. And, once
9 again, I remind the Board that I said we are dealing
10 with what the ESSA Document says and beware of the fact
11 that when we get out into a broader range of
12 literature, these terms are sometimes used in a variety
13 of ways.

14 Q. I understand.

15 A. We are talking about a relationship
16 between residues in the environment and residues in an
17 organism.

18 Now, in some cases, and particularly in
19 lab situations, it's fairly easy to measure those.
20 When we get out into a field situation residues -- you
21 can measure residues in substrates, in foliage or soil
22 or whatever it may be, and you can measure residues in
23 organisms, but they are measured in different terms.

24 Obviously a part per million, which is a
25 measurement of a weight of a pesticide to a weight of a

1 substrate, means something very different if you're
2 measuring it in a soil than it might mean if you are
3 measuring it in air.

4 Q. Could I stop you there just for a
5 moment, Mr. Kingsbury.

6 A. Mm-hmm.

7 Q. And could we back up just a step.

8 A. Yes.

9 Q. The terms bio-concentration and
10 bio-magnification were defined in the ESSA Document by
11 the authors of that report for the purposes of their
12 report; am I correct?

13 A. Right.

14 Q. And do I understand you simply to be
15 alerting those of us who read the report and the Board
16 that one has to bear the authors' definition in mind
17 when you come to read the report?

18 A. That's right.

19 Q. All right. And that that definition
20 may differ from that employed by others in other
21 endeavors?

22 A. Absolutely. Not only in the
23 literature, but in the protocols and in regulatory
24 things like the federal register.

25 Q. All right. My only point to you, Mr.

1 Kingsbury, is this that is it fair to say that the fact
2 per se of evidence of bio-concentration or
3 bio-magnification will not of and in itself prevent
4 registration or cause deregistration, if I can put it
5 that way?

6 The fact of it per se isn't enough to do
7 that, it's a much more complicated question; isn't it?

8 A. If you are dealing with the fact per
9 se to mean that at one point in time in one study one
10 shows that the concentration -- you have a value in an
11 organism that, as best you can measure, reflects a
12 higher value than what's in the environment.

13 Certainly this is an area where a number
14 of things have to be taken into account and one of the
15 major things is what happens over time.

16 Q. All right. And the point is that it
17 must be significant bio-concentration or
18 bio-magnification?

19 A. That's right.

20 Q. All right. And there is a whole
21 range of parameters that have to be looked at; is there
22 not, including things such as the toxicokinetics, the
23 rate of elimination, the excretion characteristics, the
24 metabolism characteristics, the breakdown, the time
25 frame, all of that is part of a consideration of

1 whether bio-concentration is significant or not; would
2 you agree?

3 A. Absolutely, and I could add to the
4 list with things particularly relating to the nature of
5 the exposure.

6 Q. Thank you. It has been clarified for
7 me, thank you. Could we turn then particularly to the
8 ESSA Document and I would like for the moment to deal
9 with the subject of possible impacts of pesticides in
10 forestry use situations on wildlife.

11 And as I understood your evidence with
12 respect to those types of impacts, impacts on wildlife
13 and indeed any potential impact, they were really
14 considered in two contexts; that is, the potential for
15 direct effects and the potential for indirect effects;
16 is that correct?

17 A. That's correct.

18 Q. Dealing for the moment just with
19 herbicides, I would like to review with you and ask for
20 your confirmation or clarification, as appropriate, of
21 what I understand the conclusions expressed in the ESSA
22 Document to be with respect to the impact of herbicides
23 on wildlife.

24 Am I right, first, that it was the
25 conclusion of the ESSA review team that it was most

1 unlikely that herbicides have a direct adverse toxic
2 effect on wildlife?

3 A. That's right, and I agreed with that
4 conclusion.

5 Q. All right. Am I also right that
6 following from the first conclusion it was the
7 conclusion of the ESSA review team that it was,
8 therefore, unlikely that herbicides have an adverse
9 effect on prey animals; that is, the food source for
10 carnivorous wildlife in the forest?

11 A. That's included in the conclusion,
12 but that doesn't say that some prey animals might not
13 be impacted by indirect effects.

14 Q. All right. Could I take you to page
15 63 of the ESSA Document, please.

16 A. Yes.

17 Q. Dealing with the issue of direct
18 significant toxic effects, I direct your attention to
19 the conclusion expressed at the bottom of page 63, and
20 is it fair to say in reading through the ESSA Document
21 that it was the conclusion of the review team that
22 herbicides are unlikely to have a significant toxic
23 effect on prey animals?

24 A. That is correct.

25 Q. All right. And the reviewers also

1 considered, as you point out, whether herbicides had an
2 indirect effect on wildlife and, in particular, they
3 looked at the question of possible impact on wildlife
4 habitat; am I correct?

5 A. That's correct.

6 Q. And there's quite a lengthy
7 discussion in the ESSA Document on that issue; is there
8 not?

9 A. There is.

10 Q. All right. Well, I would like to
11 deal with that issue for the moment, impact on wildlife
12 habitat, and I would like to try to summarize what I
13 understand the conclusions by the reviewers to have
14 been, and perhaps the best way to do this is actually
15 go through the report.

16 Could I ask you, if you would, to start
17 please at page 68.

18 A. Yes.

19 Q. Dealing with the first full paragraph
20 after the indented paragraphs, am I correct that the
21 authors suggest that any direct evidence of the effects
22 on wildlife of habitat changes resulting from herbicide
23 use is extremely limited?

24 A. That is what the authors report, yes.

25 Q. They also suggest; do they not, that

1 the available evidence is speculative - that's their
2 word - based on presumed changes to habitat resulting
3 from herbicide use?

4 A. That's what they report, yes.

5 Q. All right. And if we go over to the
6 neck page they suggest; do they not, and conclude or at
7 least point out that there are other forest management
8 activities apart from the use of herbicides that have a
9 far greater impact on habitat than does the use of
10 herbicides itself?

11 A. Yes.

12 Q. An example given by the ESSA
13 reviewers, for example, is the use of mechanical
14 methods to control competition?

15 A. Mm-hmm.

16 Q. Is that a yes, sir, for the record?

17 A. Yes. Sorry, yes.

18 Q. All right. Do the authors suggest as
19 well at page 69 that such effects as there are from the
20 use of herbicides on wildlife habitat are the result
21 primarily of the herbicides actually being effective
22 for their intended purpose?

23 A. That's correct.

24 Q. All right. And stated that way,
25 perhaps it is self-evident, but am I right that the

1 point of that is that one of the primary uses in
2 forestry of herbicides is for the control of competing
3 vegetation, competing plant species?

4 A. Yes.

5 Q. And, therefore, the intended effect
6 is to actually effect a change to vegetation?

7 A. Absolutely.

8 Q. Vegetation forming part of the
9 wildlife habitat in the forest?

10 A. Yes.

11 Q. So that, therefore, inherently there
12 is a change to wildlife habitat occasioned by
13 herbicides because there is a change to vegetation?

14 A. That's correct.

15 Q. All right. But that is really only
16 the preliminary step in assessing the significance of
17 the change as to whether it's positive or negative and
18 to what extent that it occurs?

19 A. It is the preliminary step and also I
20 think, as I pointed out, because there is a lack of
21 direct toxic effects, it is in fact the critical step.

22 Q. All right. And the point the ESSA
23 experts were making; is it not, is that alternatives to
24 herbicides use having the same purpose; that is,
25 control of competing vegetation, may in fact have

1 greater impacts on habitat? The alternatives may have
2 a greater impact than herbicides would?

3 A. Just clarify for me alternatives in
4 terms of...?

5 Q. Competition control. The
6 alternatives available--

7 A. Yes.

8 Q. --to the use of herbicides may have a
9 greater impact on habitat than herbicides will?

10 A. They may have a greater impact. They
11 are almost certain to have similar impacts in that if
12 in fact they are as successful at changing the plant
13 community.

14 Q. Yes, thank you. Too equally
15 effective alternatives; one being herbicides, one being
16 another forest management technique. It may well be,
17 the authors suggest, that the non-herbicidal treatment
18 may have greater effects on wildlife habitat; isn't
19 that so?

20 A. Yes. I wouldn't put great emphasis
21 on the greater. Certainly there is a -- in terms of
22 comparable, and maybe I'm just not -- I'm not sure that
23 that's supported by what is said here in terms of
24 greater effect.

25 Q. All right. Could I ask you to go --

1 not much turns on it. I thought it was a basic
2 principle, Mr. Kingsbury.

3 A. Okay.

4 Q. But could I ask you to read to
5 yourself for a moment, please, the top of page 70?

6 A. Okay. That's correct. As you have
7 pointed out, alternatives may have greater -- even
8 greater impacts on the habitat of some species.

9 Q. Thank you. And is it also not true
10 that the degree to which there is an impact by the use
11 of herbicides clearly will be a direct result of the
12 extent of the area treated and the manner of treatment?

13 A. Absolutely.

14 Q. All right. And that there are some
15 species -- depending on the home range of an animal
16 species, there may be little or no effect from the use
17 of herbicides to that species or the converse may be
18 equally true?

19 A. That's true.

20 Q. All right.

21 A. If I may, I think I'm sort of
22 thinking backwards here. I believe some of the problem
23 I had with the even greater impact is I was thinking
24 specifically of alternative tending methods but, as I
25 pointed out, I believe a number of times yesterday,

1 looking at other timber management activities, yes, I
2 would very much concur that there were even greater
3 effects from some of the other activities and, as I
4 spelled out, they are done in sequence with tending.

5 Q. All right. And am I also correct by
6 way of final summary that the ESSA authors or reviewers
7 conclude in general that herbicides, as they are used
8 now in forestry, do not have a serious -- will not
9 seriously endanger wildlife habitat; that's their
10 general conclusion?

11 A. I think that the authors have perhaps
12 not made that conclusion that they will not seriously
13 alter wildlife habitat so much as they have said that
14 they will modify wildlife habitat and that will be
15 different in terms of whether it's a positive or a
16 negative or a negligible effect depending on the
17 habitat requirements of different species of wildlife.

18 Q. Mr. Kingsbury, could I ask you to go
19 to page 71, please, to the first sentence in the first
20 full paragraph.

21 And I suggest to you, sir, that what the
22 ESSA reviewers expressly found was that, in general, it
23 would appear, based on their review, that herbicides as
24 they are used now implicitly in forestry will not
25 seriously endanger wildlife habitat?

1 A. That's what it states there, yes.

2 Q. That was their general conclusion;
3 was it not?

4 A. It certainly is written here as a
5 general conclusion in their discussion, yes.

6 Q. All right. And, in fairness to you,
7 what you have said was that you have to examine the
8 nature of the change to determine whether it's positive
9 or negative and that will vary according to species?

10 A. Yes. And I will note that in making
11 that general conclusion they have very appropriately
12 begun to talk about the things that you mentioned as
13 being important in terms of other changes and the size
14 and scale of herbicide-induced changes.

15 Q. All right. And the authors then go
16 on - I don't propose to go through each example, Mr.
17 Kingsbury - but the authors then go on to point out a
18 number of situations where habitat changes in fact
19 effect positive changes to habitat; isn't that so?

20 A. Very much so, yes.

21 Q. All right. And one of the issues or
22 changes much discussed in this report is the effect on
23 browse for moose and deer?

24 A. Yes.

25 Q. And a number of reports are referred

1 to by the ESSA reviewers which suggest that there are
2 either -- which suggest that there is a beneficial
3 impact from the use of herbicides on deer and moose
4 browse materials; isn't that so?

5 A. At some points in the time cycle of
6 blank community response, yes.

7 Q. All right. Well, for example, the
8 reviewers refer to an article published by Sullivan, et
9 al, in 1985 which demonstrated a preference by deer for
10 glyphosate-treated browse; isn't that so?

11 A. They did cite that study; yes, that's
12 correct.

13 Q. Am I correct, Mr. Kingsbury, that
14 that study is contained in a textbook entitled: The
15 Herbicide Glyphosate, published by Butterworth in 1985?

16 A. That's right, and in fact there is a
17 chapter in that book authored by Sullivan.

18 Q. And it's from that chapter that the
19 reference is made, or with respect to that chapter;
20 isn't that the case?

21 A. That's correct, and it's also
22 published in Canadian Journal of Zoology, I believe.

23 Q. All right. Are you familiar with
24 this textbook, Mr. Kingsbury?

25 A. I'm familiar specifically with the

1 portions of that textbook relating to impacts on
2 organisms and I'm also familiar, to a lesser extent,
3 with other parts of the book, yes.

4 Q. All right. Are you familiar with the
5 portion in the text dealing with the state and
6 biological consequences of glyphosate in the aquatic
7 environment?

8 A. Yes.

9 Q. All right. Would you agree with me
10 or can you tell me whether this book is recognized as
11 an authoratitive text on the subject of the herbicide
12 glyphosate?

13 A. I believe it's one of the most recent
14 and comprehensive reviews of the material, yes.

15 Q. Thank you. Then apart from the
16 Sullivan work which is referred to in the ESSA Document
17 on this issue of impact on browse for moose and deer,
18 the ESSA reviewers suggest with and support their
19 suggestion with reference to a number of studies that
20 better foliage may be available following herbicide
21 treatment as new growth is introduced; isn't that so?

22 A. Yes.

23 Q. All right. And one of the articles
24 referred to in the ESSA Document at page 74 in support
25 of that suggestion -- well, there are several. One is

1 by Sullivan, 1985, we have already discussed that. One
2 is by Crefting and Hanson, 1969; am I correct?

3 A. Could you just...

4 Q. Second paragraph, page 74.

5 A. Second paragraph, okay. Yes, that's
6 correct.

7 Q. There are a number of authorities
8 referenced there?

9 A. Yes.

10 Q. A number of studies, Crefting and
11 Hanson 1969, Baroco, et al, 1972. Do you see that?

12 A. Yes.

13 Q. Sullivan and Sullivan, 1980
14 unpublished data; do you see that?

15 A. Yes.

16 Q. And the fourth is an article by
17 Newton, et al which is described as being in press; is
18 that correct?

19 A. That's yes.

20 Q. I'm going to show you an article, Mr.
21 Kingsbury, a copy of which I earlier provided to you
22 entitled: Fate of Glyphosate. Actually that is the
23 wrong article, excuse me. Could I just have a moment,
24 please.

25 Entitled: Browse Availability After

1 Conifer Release in Maine's Spruce/Fir Forests by
2 Michael Newton and four other authors including an M.
3 L. McCormick, Jr. Are you familiar with the article?

4 A. Yes.

5 Q. Is this the article that is referred
6 to in the ESSA Document as being in press?

7 A. That's my belief. Yes, that it is.

8 MS. CRONK: That will be the next
9 exhibit, please.

10 THE CHAIRMAN: Exhibit 722.

11 ---EXHIBIT NO. 722: Article entitled: Browse
12 Availability After Conifer Release
13 in Maine's Spruce/Fir Forests by
14 Newton, et al.

14 MS. CRONK: Q. This article, as I
15 understand it, Mr. Kingsbury, is to be published. If
16 it was not published in July of this year, it's to be
17 published in August of this year in a journal entitled:
18 The Journal of Wildlife Management, a U.S. publication.
19 Is that your understanding?

20 MR. KINGSBURY: A. The article is in
21 fact published.

22 Q. It is published.

23 A. With the citations there.

24 Q. And I take it that the ESSA reviewers
25 had available to them a pre-print copy of the document?

1 A. Yes.

2 Q. All right. And I would refer you to
3 the abstract reference to the paper. Am I correct that
4 in this particular study a variety of herbicides,
5 including glyphosate, were applied seven years after
6 harvest to a spruce/fir forest in the northeast United
7 States?

8 A. That's correct.

9 Q. And according to the abstract
10 description of results, the study indicated, as a
11 result of the application of glyphosate that a four to
12 eight fold increase in available browse resulted?

13 A. That's correct.

14 Q. And, further, that nearly all plant
15 species present before the herbicide treatment remained
16 some nine years later with a greater percentage of
17 sungrown foliage and twigs than existed in the
18 untreated plots; is that correct?

19 A. That's correct.

20 Q. In essence, the study suggests that
21 nine years after treatment of a forestry site with
22 glyphosate, browse on the sprayed plots was
23 significantly better than on the untreated plots; isn't
24 that so?

25 A. Not only that, but it I believe

1 demonstrates that for sites sprayed with a variety of
2 herbicide materials.

3 Q. Including...?

4 A. Including glyphosate.

5 Q. Yes, in the phenoxy herbicide
6 category; am I correct?

7 A. Right.

8 Q. All right. And if we go to page 644
9 very briefly in the first full paragraph under Table
10 1--

11 A. Mm-hmm.

12 Q. --I suggest that the nature and the
13 purpose of the study is outlined and it's indicated as
14 outlined in the abstract that:

15 "Several herbicides in the phenoxy
16 category were experimentally applied for
17 conifer release seven years after harvest
18 with the objective specifically of
19 recording vegetation development that
20 followed and to investigate and relate
21 that development to browse availability."
22 That was the purpose and the nature of
23 the study; is that correct?

24 A. Yes.

25 Q. And, in general terms, just to move

1 through it quickly if we can, am I correct that the
2 results were analysed one year after treatment and
3 again nine years after treatment?

4 A. Yes.

5 Q. And am I correct that the study
6 supports the earlier literature referred to in the ESSA
7 Document that browse may be improved following
8 herbicide application if one of the herbicides is in
9 the phenoxy group?

10 A. Yes.

11 Q. And the conclusions by the authors
12 are set out over at page 648. I would ask you to go
13 there for a moment, if you would.

14 I direct your attention to the very last
15 paragraph. Can you confirm for me, Mr. Kingsbury, that
16 apart from the conclusions you have already indicated
17 to the Board were reached by the authors, there is also
18 an indication by the authors that, to the best of their
19 knowledge, none of the herbicide treatments eradicated
20 any plant species?

21 A. That's correct.

22 Q. Further, the tall cover was not
23 eliminated rather simply reduced with ratios of cover
24 types changed?

25 A. That's correct.

1 Q. All right. I take it from that that
2 all plant species remained following treatment although
3 the compositions of the plant species or the extent of
4 each plant species may have differed? Is that a fair
5 reading of it?

6 A. That's the way I would read it, yes.

7 Q. All right. Do you know Professor
8 Michael Newton either by reputation or personally?

9 A. Yes.

10 Q. What is his position or involvement
11 in forestry?

12 A. He's had a longstanding involvement
13 in forestry field trials in the United States.

14 Q. Is he an acknowledged expert in your
15 opinion concerning matters related to forest management
16 practice and, in particular, the use of pesticides with
17 particular emphasize on herbicides?

18 A. Yes.

19 Q. And are you -- do you know either
20 personally or by reputation Professor M. L. McCormick
21 Jr., of the College of Natural Resources, University of
22 Maine, Orno?

23 A. Yes, I do.

24 Q. And, again, what is the area of his
25 involvement in forestry?

1 A. Once again, considerable and
2 extensive experience within the area of herbicide use
3 and applications and the effects in forestry
4 situations.

5 Q. And do you regard him as an expert in
6 those areas?

7 A. Yes, I do.

8 MS. CRONK: I am sorry, sir, I neglected
9 to know the exhibit number for that exhibit.

10 THE CHAIRMAN: It was 722.

11 MS. CRONK: Thank you.

12 THE CHAIRMAN: Mr. Kingsbury, comparisons
13 have been made in some of this literature between the
14 use of herbicides and other man or human treatments in
15 terms of forestry practices; site preparation,
16 mechanical and otherwise, this kind of thing.

17 What about comparisons between the change
18 in or effect on habitat of some natural occurrences
19 such as fire vis-a-vis the impact on changes in habitat
20 due to fire as opposed to impact or changes on habitat
21 due to the use of pesticides?

22 I guess what I'm getting at here: The
23 areas affected would be infinitely greater in terms of
24 habitat affected by non-human interference with the
25 forest.

1 MR. KINGSBURY: I guess --

2 THE CHAIRMAN: Most of the comparisons
3 seem to be made with human interferences.

4 MR. KINGSBURY: That's correct and I
5 guess -- I believe you probably mentioned the critical
6 aspect of the non -- the natural events that impact on
7 wildlife habitat in the forests such as fire, then of
8 course pest and disease activities, is that their
9 occurrence is something that, in terms of a spacial and
10 a temporal fashion, it happens when it happens and
11 wildlife simply has to respond.

12 And if it's a large fire, obviously, it
13 can have a very negative effect over a very large area,
14 or if it's something like, you know, a historical
15 budworm infestation as we know from the forest record,
16 prior to human intervention into that that vast areas
17 may all of a sudden drastically change in terms of the
18 wildlife habitat that they preserve.

19 THE CHAIRMAN: So could you reasonably
20 conclude that whatever the changes might be due to the
21 use of herbicides, and even some of those changes on
22 habitat, may be positive in the long run--

23 MR. KINGSBURY: Yes.

24 THE CHAIRMAN: --they would be minor in
25 terms of amount of habitat affected by including, in

1 addition to human interference, natural interference?

2 MR. KINGSBURY: Absolutely.

3 THE CHAIRMAN: Or naturally-induced
4 changes to habitat?

5 MR. KINGSBURY: That's correct.

6 THE CHAIRMAN: Thank you.

7 MS. CRONK: Q. Mr. Kingsbury, as I
8 understand it, one of the other very relevant issues in
9 the whole consideration of the assessment of risk from
10 the use of herbicides is the issue of the persistence
11 in and effect of herbicides on soils and water as well
12 as directly on animals; am I right?

13 MR. KINGSBURY: A. That's correct.

14 Q. All right. And in terms of direct
15 effect on animals, direct impact on wildlife, we have
16 already reviewed what the ESSA team of experts
17 concluded in that regard?

18 A. Yes.

19 Q. Am I also correct that the data
20 requirements of relevant federal regulatory agencies in
21 this country require specific studies to be prepared
22 and submitted in support of a registration application
23 dealing with such issues as degradation of a pesticide
24 in soils, mobility, persistence not only in soil but
25 also in water, in sediments?

1 A. Not only specific studies -- I'm not
2 sure whether you have included, a petition for forestry
3 registration would in fact require data of that type
4 specific to the forestry sites and use patterns
5 intended.

6 Q. All right. And that is a specific
7 requirement I believe of Environment Canada as one of
8 the involved reviewing agencies; isn't that so?

9 A. That's correct.

10 Q. All right. And as well the data
11 regarding the specific metabolism of the chemical is
12 required?

13 A. Yes.

14 Q. Are you familiar, Mr. Kingsbury, with
15 an article published in 1984 by Professor Newton to
16 whom you referred a few moments ago together with
17 others entitled: The Fate of Glyphosate in an Oregon
18 Forest Eco-system?

19 A. Yes, I am.

20 THE CHAIRMAN: Exhibit 723.

21 MS. CRONK: (handed)

22 ---EXHIBIT NO. 723: Article entitled: The Fate of
23 Glyphosate in an Oregon Forest
24 Eco-system by Newton, 1984.

25 MS. CRONK: Q. Mr. Kingsbury, for our

1 ease of discussion with respect to this document, could
2 I ask you just for a moment to number each of the
3 pages. They are numbered from the publication but many
4 of them didn't come out in the photocopying and I have
5 it that there is eight pages, one being the cover page.

6 MR. KINGSBURY: A. Done.

7 Q. All right. Mr. Kingsbury, it is my
8 understanding that this article by Professor Newton and
9 his colleagues is regarded really as a classic
10 published study of the investigation of a particular
11 herbicide in a forest eco-system, particular in this
12 case to glyphosate, but the study itself is well known
13 and is considered a classic in its field. Would you
14 agree with that?

15 A. Yes, I would.

16 Q. All right. The purpose of the study
17 and the study objectives are set out on page 1 and I
18 would refer you in this regard to the second -- well,
19 the first paragraph above the section entitled:
20 Experimental section. Do you see that?

21 A. Yes.

22 Q. And am I correct that the study
23 objectives, as outlined by the authors, were
24 specifically to determine first glyphosate deposits in
25 various strata of forest vegetation after aerial

1 application and its persistence in those strata and in
2 litter and in soil."

3 A. Yes.

4 Q. That was one of the objectives?

5 A. That's right.

6 Q. And, secondly, to determine
7 glyphosate concentrations in stream water, sediment and
8 fish after direct application to open streams?

9 A. That's correct. It's direct
10 application in the context that we discussed it, as
11 aircraft directly flying over the aquatic system being
12 studied.

13 Q. All right. And, thirdly, the third
14 objective was to determine glyphosate exposure and
15 retention levels in various forest mammals?

16 A. That's correct.

17 Q. And, finally, the occurrence and
18 persistence of the major metabolite of glyphosate known
19 as AMPA, that is the acronym for it?

20 A. That's right.

21 Q. And to determine the occurrence and
22 persistence of what is known as NNG a trace impurity in
23 glyphosate; am I correct?

24 A. That's correct.

25 Q. All right. And, again, in an effort

1 to summarize the nature of this study and the work that
2 was done, am I correct that there were two study sites
3 involved; the first is as described by the authors as
4 being the primary study site and the second, logically
5 enough, the secondary study site?

6 A. That's correct.

7 Q. And am I correct that the first site
8 was an eight hectare unit situate in Oregon over which
9 a small stream flowed through the site lengthwise and
10 on which existed two large shallow beaver ponds?

11 A. That's right.

12 Q. Apart from other topographical
13 features.

14 A. Mm-hmm.

15 Q. And am I correct that the study sets
16 out both the nature of the pre-treatment sampling, the
17 nature of the treatment application, meaning the way
18 the glyphosate was applied, and also the nature of the
19 post-treatment sampling that was carried out?

20 A. That's right.

21 Q. And would you agree with me that both
22 the pre-treatment and the post-treatment sampling were
23 considerable for the purposes of this study?

24 A. Yes.

25 Q. Dealing just with the pre-treatment

1 side of it, the article suggests that samples were
2 taken of vegetation, of soil and of -- of soil both
3 covered and uncovered and of litter before any
4 application of glyphosate?

5 A. Yes.

6 Q. Stream water was also sampled as were
7 various terrestrial animals prior to the application of
8 any herbicide?

9 A. Yes.

10 Q. And by the sampling of various
11 animals they essentially collected various specimen
12 species types from the site before the application of
13 glyphosate?

14 A. Yes.

15 Q. And then two loads of glyphosate were
16 applied at the rates described on page 2 as we have
17 numbered them? There were two applications of
18 glyphosate? I'm looking under treatment applications.

19 A. Yes. Just to clarify, when it says
20 two loads, I believe that is two helicopter loads, that
21 in fact.

22 Q. One application then?

23 A. There was one application. The
24 treatment rate was 3.3 kilograms per hectare and I just
25 note that. I believe that is about one and a half

1 times the maximum rate registered in Canada.

2 Q. All right, thank you. So there was
3 one application at rates in excess of what would be
4 permitted here?

5 A. Yes.

6 Q. All right. And then post-treatment,
7 again, a number of specimens were taken and am I
8 correct they included both aquatic and terrestrial
9 samples?

10 A. Yes, and a number of substrates as I
11 described that in both of those categories.

12 Q. And, in addition, both terrestrial
13 and animal samples were collected actually at the time
14 of spraying and at set intervals thereafter?

15 A. Yes.

16 Q. And litter samples, water samples and
17 stream sediment were sampled?

18 A. Yes.

19 Q. Then, the secondary study site, as I
20 understand it, was introduced to the study largely
21 because shortly after the application of glyphosate on
22 the primary site there had been a very unanticipated
23 rainfall?

24 A. Yes.

25 Q. And they had 21 millimetres of rain

1 within 36 hours, as I understand it?

2 A. That's right.

3 Q. And that gave rise to the possibility
4 of washoff?

5 A. One would think so, yes.

6 Q. A lot of rain. And, therefore, the
7 second site also in Oregon was selected and aerial
8 application at the same rate of glyphosate and the same
9 volume of delivery with the same pilots and same
10 equipment was used?

11 A. Yes.

12 Q. All right. And then at the bottom of
13 page 3 we find the detailed description set out of the
14 results obtained from both sites during the course of
15 the study and over at page 6, as we have numbered it,
16 on the bottom of the left-hand column we find the
17 beginning of the discussion; is that correct?

18 A. Yes.

19 Q. All right. Could I ask you first, if
20 you would, to outline for the Board what conclusions
21 were formed by the authors based on their work
22 regarding the metabolism of glyphosate, and I would
23 refer you in that regard to the discussion beginning
24 the first two sentences at the bottom of the column on
25 the left on page 6?

1 A. Sorry, under metabolites and
2 contaminants.

3 Q. Sorry, under discussion.

4 A. Yes, okay.

5 Q. Could you indicate to the Board what
6 the observations or conclusions were of the authors
7 regarding the metabolism of this chemical based on this
8 study?

9 A. Okay. The authors concluded that
10 glyphosate disappeared rapidly from this forest system,
11 that it did not move into water from soil and, in fact,
12 that much of the material degraded in the substrates
13 where it landed which, in this study, was primarily in
14 the vegetation.

15 Q. All right. And are those findings
16 consistent with your understanding regarding the
17 metabolism of glyphosate?

18 A. Very much so, yes.

19 Q. All right. And I direct your
20 attention as well to the observations made at the
21 bottom of page 6 in the right-hand column which read:

22 "The findings of absorbed glyphosate in
23 stream sediment are of interest to the
24 interpretation of soil data."

25 A. Yes.

1 Q. "The ability of sediments to glean
2 moderate amounts of glyphosate from water
3 when those sediments have much higher
4 glyphosate concentrations than water
5 corroborates earlier reports that
6 particulate matter has a high absorptive
7 capacity for glyphosate."

8 A. Yes.

9 Q. Do you see that? Now, stopping there
10 for a moment, Mr. Kingsbury. Again, from my
11 perspective to try to approach it from a layman's
12 perspective, I understand that what this study
13 demonstrated as a result of these field tests was that
14 glyphosate, when it metabolized, in fact was a
15 attracted to and absorbed to sediment, to particulate
16 matter in water and did so in a way which led the
17 authors to conclude that it does not -- in fact that
18 its mobility in water was low.

19 Is that a fair summation of what the
20 implications of those observations were?

21 A. I believe that you indicated when it
22 metabolized that it basically became bound to sediments
23 in water.

24 My interpretation is that the glyphosate
25 itself, not simply glyphosate that had been

1 metabolized, i.e., undergone some degradation, became
2 strongly attached to the sediment in the aquatic
3 systems.

4 Q. Apart from that amplification, is the
5 rest of what I suggested to you a fair--

6 A. Yes.

7 Q. --and accurate reflection of what the
8 study found.

9 A. Yes.

10 Q. I'm sorry?

11 A. Yes.

12 Q. That has; does it not, implications
13 for the issue of the potential for contaminant
14 transport of this chemical in water?

15 A. Yes, it does.

16 Q. And as to the potential for a
17 contamination of water sources both surface and
18 groundwater?

19 A. It would imply that such
20 contamination is dramatically limited.

21 Q. All right. And I would ask you in
22 that regard if you would, please, over on page 7 to
23 look at the further observations of the authors in this
24 regard. And I'm still looking at the first paragraph
25 at the top of the page there where the authors

1 indicate:

2 "We did not identify retention by
3 sediment soil fraction. Should retention
4 in sediments be studied intensively,
5 techniques for separating soil into
6 organic and inorganic compounds would be
7 needed. Nevertheless, the chemical
8 showed a high affinity for solids even
9 when clean water was running through in
10 substantial excess. The finding of
11 roughly comparable AMPA levels in
12 sediments and uplands soils suggests that
13 residues are virtually immobile while
14 being metabolized even in the presence of
15 substantial leaching opportunity."

16 Does that support the suggestion I made
17 to you a few moments ago, Mr. Kingsbury, that the
18 findings of this study have direct implications for the
19 potential contaminant transport of glyphosate in water?

20 A. It is in fact the findings on which
21 that contention is supported.

22 Q. All right. And they are indicating;
23 are they not, that even in the presence of clean
24 running water the characteristics of the chemical were
25 such that it bound to sediment, to soil particles as

1 distinct from being attracted to water, if I can put it
2 that way, moving through the water?

3 A. That's right. Even when clean
4 flowing water was running by, the material clung to the
5 sediments.

6 Q. All right. And still dealing with
7 the metabolism characteristics of the chemical, could I
8 ask you to look to the last paragraph in the right-hand
9 column on page 7 which indicates:

10 "Rates of degradation observed in foliage
11 and soil corroborate the manufacturer's
12 general statement based on agricultural
13 data that glyphosate is broken down
14 quickly."

15 Stopping there. Is that your
16 understanding of the study results?

17 A. Yes.

18 Q. And do you accept them as being
19 reliable in the face of the results that they obtained?

20 A. Yes.

21 Q. And further the authors indicate:

22 "Conditions of degradation here are
23 likely to have been quite different from
24 those encountered in agricultural studies
25 suggesting a relative insensitivity of

1 glyphosate degradation to environment
2 influence and a tendency toward rapid
3 disappearance from both simple and
4 complex eco-systems."

5 Do you agree with that conclusion?

6 A. Yes.

7 Q. And what do you understand the term a
8 relative insensitivity of glyphosate degradation to
9 environmental influence to mean?

10 A. I would interpret that to mean that
11 the degradation of glyphosate is not based on a very
12 narrow range of parameters; that in fact there are
13 broad pathways that tend to operate in virtually all
14 simple or complex eco-systems that would break this
15 material down.

16 Q. And then dealing with the -- dealing
17 further with the implications of the study for the
18 movement and effect of glyphosate on water resources,
19 could I ask you to go back to the left-hand column on
20 page 7.

21 A. Yes.

22 Q. And look specifically this time at
23 the first full paragraph beginning with the words:

24 "The absence of glyphosate..."

25 Do you see that?

1 A. Yes.

2 Q. All right. And am I correct that the
3 suggestion made by the authors in that paragraph and
4 the immediately following paragraph is that based on
5 the tendencies of the chemical which they observed,
6 that contamination of water -- well, first, they found
7 that any contamination of water by direct spray
8 application was within the range of safety to stream
9 plants and irrigated crops suggested in the scientific
10 literature. They did not find excess contamination?

11 A. That's right.

12 Q. And, further, am I correct that they
13 concluded based on the data which they actually
14 measured from these tests that there was no mutual risk
15 from including forest streams within the spray pattern
16 as a result of using this chemical?

17 A. That's right.

18 Q. And, further, that there was no
19 effect on water quality from devegetation resulting
20 from the spraying until the following year?

21 A. That's right.

22 Q. At which time they indicate dead
23 shade would persist?

24 A. That is their conclusion, yes.

25 Q. All right. That conclusion speaks to

1 the issue of aquatic habitat; does it not, vegetation
2 on the shores?

3 A. Indirect effects, yes.

4 Q. All right. And what is the
5 implications of saying that a year later when effects
6 were observed dead shade would persist?

7 A. They are basically saying that the
8 modification of the aquatic habitat at that time would
9 be limited, because under -- in this site and the way
10 they treated it there would still be considerable cover
11 over the stream from vegetation that had been killed
12 but which still presented a barrier to sunlight
13 penetration. That is the way I would interpret it.

14 Q. And then, Mr. Kingsbury, they
15 conclude finally; do they not, based on the data which
16 they collected and their observations that they
17 anticipated negligible effect overall of glyphosate on
18 forest water quality? That was their conclusion; was
19 it not?

20 A. That's correct.

21 Q. And do you their share their view
22 based on the data evidenced in this report?

23 A. I would say that in this report they
24 are anticipating an effect, that the study in itself is
25 not sufficient to provide proof of that effect, but I

1 would say that they have gone a very considerable
2 distance to eliminating probable causes of the effect.

3 Q. And do you support the directions
4 suggested by their conclusion and; that is, that their
5 data supports the view that it is unlikely that there
6 would be significant negative effect to forest water
7 quality from the use of glyphosate?

8 A. I would very much do that. Of
9 course, I have the advantage of hindsight in that other
10 studies, and thinking particularly of the Carnation
11 Creek experiment, which would have directly generated
12 data on many of the things that contribute to the
13 direction they are talking about confirms that that, in
14 fact, has largely been the case.

15 Q. All right. So subsequent research
16 corroborated the suggestion they were making back in
17 1984 based on their data?

18 A. That's correct.

19 Q. All right. And do you then agree
20 today with the conclusion that they are suggesting in
21 this paper; that being, that there will be negligible
22 effect overall of glyphosate on forest water quality in
23 the circumstances of an aerial application of this
24 kind?

25 A. That's correct.

1 Q. All right. The study also
2 considered; did it not, the impact on wildlife of the
3 use of glyphosate on both of these study sites?

4 A. Yes, it did.

5 Q. And am I correct that, if I could put
6 it in general terms, that based on the data which they
7 collected, they concluded that the toxicological risk
8 from forest use of glyphosate was probably, in their
9 terms, zero for either wildlife or humans?

10 In essence, they were saying there was
11 very negligible risk?

12 A. That is their conclusion, yes.

13 Q. All right. And they based that
14 conclusion; did they not, on their observation of low
15 exposures and rapid elimination of the chemical in the
16 animals that they sampled?

17 A. Yes.

18 Q. And that was so notwithstanding or
19 regardless of any feeding preferences of the wild
20 animals they tested?

21 A. That's correct.

22 Q. Would you agree with the suggestion
23 made by Professor Newton and his colleagues that those
24 two features afford reassuring evidence that wildlife
25 and their food supplies are unlikely to be threatened

1 by glyphosate?

2 A. Yes.

3 Q. The fact that glyphosate is rapidly
4 eliminated is particularly significant in that context;
5 is it not?

6 A. Certainly, as we discussed in talking
7 about direct effects in toxicology just as environment,
8 once you no longer have exposure you no longer have
9 possibility of effect. So, yes, I agree the fact that
10 it rapidly disappears and eliminates potential for
11 further effect, direct effect.

12 Q. Well, in fact, just as an example of
13 that principle and conclusion, the authors indicate
14 that with the small mammals that they tested,

15 "...detectable intake of the chemical was
16 limited to the first month after
17 treatment and the elimination systems
18 maintained body levels low enough to
19 suggest that the non-visceral parts were
20 a part of the elimination pipeline."

21 A. That's correct.

22 Q. I am quoting from the article.

23 A. Yes.

24 Q. And, in fact, when you look at the
25 actual residues which they measured following treatment

1 the highest contents were in the stomach; were they
2 not?

3 A. Yes.

4 Q. That suggests direct ingestion?

5 A. Yes.

6 Q. As opposed to accumulation in the
7 body?

8 A. That's right.

9 Q. So, in essence, if I can summarize
10 that, as I understand, the data which the authors
11 collected demonstrates and supports, first, a rapid
12 elimination rate of the chemical from animal systems?

13 A. Yes.

14 Q. Secondly, a negligible effect on
15 water quality?

16 A. Yes.

17 Q. Thirdly, a rapid degradation pattern?

18 A. Yes.

19 Q. Fourthly, an infinity of the chemical
20 in terms of its metabolism for sediment as distinct
21 from water?

22 A. Yes.

23 Q. And that there -- and, fifthly,
24 negligible toxicological risk to either wildlife or
25 humans from the use of the chemical?

1 A. That's right.

2 Q. And, finally, with respect to this
3 study, you will recall that one of the objectives, the
4 stated objectives was to detect the occurrence and
5 persistence of what I termed NNG which, as I understand
6 it, is nitrosylglyphosate - am I pronouncing that
7 correctly? In any event, it is a trace impurity in
8 glyphosate?

9 A. That's my understanding, yes.

10 Q. It is known as NNG?

11 A. Yes.

12 Q. And, further, to similarly detect the
13 occurrence and persistence of AMPA, the major breakdown
14 or the major identifiable metabolite of glyphosate?

15 A. That's correct.

16 Q. And if we look over at page 6 of the
17 study, would I be accurate in suggesting that the
18 scientists failed to find any AMPA in any animal tissue
19 after 14 days following herbicide treatment?

20 Sorry, I am looking at page 6 the column
21 on the left.

22 A. Yes.

23 Q. It is paragraph --

24 A. I'm just... That's correct.

25 Q. All right. And they concluded from

1 that that the chemical was eliminated rapidly and
2 absorbed in negligible amounts?

3 A. Yes.

4 Q. All right. And with respect to NNG,
5 am I correct that none of the fish collections which
6 they tested showed measurable whole body levels of
7 glyphosate, AMPA or NNG at a detection limit of 0.05 -
8 is that milligrams per kilogram of body weight -

9 A. That's correct.

10 Q. --despite glyphosate concentrations
11 in stream water that remained in the detectable range
12 for three days?

13 A. That's correct.

14 Q. All right. In essence they didn't
15 find any NNG of concern; is that correct?

16 A. In fish they failed to show NNG or
17 AMPA or glyphosate.

18 Q. All right. And the AMPA that they
19 found in other species and animal tissues was found to
20 eliminate very rapidly; is that correct?

21 A. Yes.

22 Q. Mr. Kingsbury, that's been a rather
23 long way of reviewing this article, but would you agree
24 with me if I suggested to you that the data presented
25 in this paper, coupled with the later research outlined

1 in the ESSA Document, confirms that there is very
2 little, if any, environmental risk afforded in the
3 forest eco-system to the use of glyphosate in forestry
4 applications?

5 A. In terms of the direct toxilological
6 risks and the risks that come from persistence of
7 glyphosate residues in the environment, yes, I would
8 agree.

9 Q. All right.

10 A. I would still note that there are
11 indirect effects through the desired action of
12 glyphosate that for some species, at some point, might
13 be considered by some people to be negative effects.

14 Q. I wasn't suggesting there was no
15 effect at all, but rather that it was not adverse in
16 terms of environmental impact?

17 A. That's correct.

18 Q. All right. And with respect to the
19 issue of glyphosate in fish, I asked you earlier if you
20 were familiar with the chapter in the Grosbard and
21 Atkinson text entitled: The Herbicide Glyphosate that
22 deals specifically with the effect of glyphosate on
23 aquatic organisms, and I believe you told me you were?

24 A. Yes.

25 Q. I would like to read a short passage

1 of that text to you and ask you whether you agree or
2 disagree with it, Mr. Kingsbury.

3 I am reading from page 216 in the
4 conclusion section of this chapter. It reads as
5 follows:

6 "Glyphosate, when used as recommended by
7 the manufacturer, is unlikely to enter
8 water courses through run-off or leaching
9 following terrestrial application.

10 Clearly, significant residues could enter
11 water when Roundup is used as an aquatic
12 herbicide."

13 Stopping there for a moment, that's if it
14 is applied directly into the water?

15 A. That's correct, and...

16 Q. "In static water, residues decline
17 fairly rapidly due mainly to absorption
18 onto particulate matter. In some
19 flowing water the conditions could be
20 such that glyphosate could remain in
21 solution or absorbed onto particulate
22 matter in suspension for some distance
23 downstream from the point of application.
24 Although the rate of degradation of
25 residues depends on pH, temperature and

1 the presence of micro-organisms, most
2 aquatic environments likely to receive
3 Roundup treatment will provide the most
4 ideal conditions for degradation."
5 Stopping there. Do you agree with that?

6 A. Yes.

7 Q. "The field experience seems to
8 support the laboratory predictions that
9 residues will be absorbed and will
10 eventually break down."

11 Do you agree or disagree with that?

12 A. I agree.

13 Q. That is in fact consistent with the
14 Newton study; is it not?

15 A. And other studies done subsequently,
16 yes.

17 Q. "Therefore, it is unlikely that
18 glyphosate will affect aquatic organisms
19 at the concentrations found in the
20 environment after use at the recommended
21 rates."

22 Do you agree?

23 A. That's correct.

24 Q. And finally:

25 "It is also unlikely that residues will

1 be accumulated in fish tissues."

2 Do you agree?

3 A. I agree.

4 Q. All right.

5 MS. CRONK: And then finally, Mr.

6 Chairman, I would like to mark as the next exhibit
7 OFIA -- the response to OFIA Interrogatory No. 28T from
8 Panel 13 which is a paper entitled: The Determination
9 of Persistence, Movement and Degradation of Hexazinone
10 in Selected Canadian Boreal Forest Soils.

11 THE CHAIRMAN: Exhibit 724. Ms. Cronk,
12 can you indicate how much longer you might be?

13 MS. CRONK: I would expect to be ten
14 minutes, sir.

15 THE CHAIRMAN: Okay. We will finish off
16 then. Thank you.

17 MS. CRONK: (handed)

18 THE CHAIRMAN: Thank you.

19 ---EXHIBIT NO. 724: Paper entitled: The Determination
20 of Persistence, Movement and
21 Degradation of Hexazinone in
Selected Canadian Boreal Forest
Soils.

22 MS. CRONK: Q. Are you familiar with
23 this paper, Mr. Kingsbury?

24 MR. KINGSBURY: A. Yes, I am.

25 Q. It considers in the context of

1 hexazinone the same persistence, mobility, metabolism
2 and degradation issues as were relevant to the Newton
3 paper on glyphosate; is that so?

4 A. It deals with a narrower range,
5 primarily the issues of persistence, mobility in forest
6 soils.

7 Q. And one of the authors of this report
8 was Robert Campbell who has previously testified in
9 these proceedings?

10 A. That's correct.

11 Q. All right. What was the general
12 nature and purpose of the study? Can you help me with
13 that?

14 A. This was a soil -- or a study that
15 was carried out under the impetus of the Institute I
16 worked for to look at the effects in two different
17 types of soil, in boreal situations in Ontario
18 specifically, and for those two types of soil which
19 were basically a clay and a sand site to see, (1) how
20 hexazinone that was applied directly to that soil would
21 persist, to see whether it would move both vertically
22 downward within the soil profile and also horizontally
23 down a slope after application.

24 Q. All right. And dealing with the
25 results of the study, let's deal with mobility first as

1 you mentioned it last.

2 Can you outline for the Board what the
3 conclusions were, based on the evidence presented in
4 this test, regarding the mobility characteristics of
5 this chemical?

6 A. The conclusions are basically that
7 the material showed very little movement downward into
8 the soil column and there was no indications of
9 movement laterally. That reflected the fact that the
10 first sampling point was, I believe, one or three
11 metres from the edge of the treated area -- three
12 metres, so it did not move three metres.

13 Q. And what were the conclusions based
14 on the data emerging from this study regarding the
15 leaching properties of this chemical?

16 A. Basically that there was very limited
17 leaching potential for this chemical on either sand or
18 clay sites.

19 Q. And, in your opinion, was that
20 supported by the data which emerged from this study?

21 A. Absolutely.

22 Q. All right. And what were the
23 conclusions reached regarding the degradation
24 characteristics of the chemical?

25 A. That in fact the material showed a

1 degradation to -- to a half life, as we discussed it
2 yesterday, in the order of 43 days in both sites and
3 that the material continued to degrade from that time
4 onward to very low levels.

5 Q. All right. Rapid degradation then?

6 A. It certainly is a -- rapid is a
7 relative term. Certainly a comparable degradation rate
8 to what is known for herbicides used in -- other
9 forestry herbicides and herbicides in general in soils.

10 Q. And what were the conclusions of the
11 study regarding the persistence of the chemical, or
12 have you dealt with that in the context of talking
13 about degradation?

14 A. Basically that it would not persist
15 within these soil types, that it would in fact degrade.

16 Q. And do you agree or disagree with the
17 conclusions expressed in this study, Mr. Kingsbury?

18 A. I would agree.

19 Q. Thank you. Mr. Kingsbury, there was
20 just one other matter and it is really by way of
21 clarification of what I understand your concluding
22 remarks to have been to the Board.

23 I have consulted with my colleague Mr.
24 Shibitani because we don't have a transcript to see
25 whether his notes and mine were the same.

1 I understood you to tell the Board
2 yesterday at the conclusion of your evidence-in-chief
3 that it was your opinion, and I am quoting:

4 "The pesticides registered for use in
5 forest management have negligible
6 or limited impact on ecologically
7 critical aspects of sensitive non-target
8 communities."

9 Was that what you said to the Board? Was
10 that your evidence?

11 A. I believe that's exactly what I said
12 to the Board.

13 Q. All right. You will forgive me, Mr.
14 Kingsbury, if I observe as a layperson that that's a
15 rather complicated statement. I wish to ensure before
16 your evidence is complete that I understand exactly
17 what it is that you mean by that.

18 Do you mean by that statement that the
19 pesticides that we are concerned with in this
20 proceeding, as used in forestry applications in the
21 forest, are safe for use in ecological terms?

22 A. I think that I am, in general,
23 concurring with that in that I believe the way I have
24 stated it is that using the assessment strategy that we
25 employ to assess them we find that there are, in fact,

1 negligible or limited effects on the environment.

2 Q. And the parameters that you look to
3 for assessment are those which are perceived to be most
4 sensitive?

5 A. And they are the parameters that we
6 believe are most reflective of overall effects on the
7 environment, yes.

8 Q. And, as well, those that are
9 considered to be the most sensitive?

10 A. Yes. In fact, that's one of the
11 reasons we select those parameters.

12 Q. All right. Is it then your opinion,
13 Mr. Kingsbury, that the pesticides in use or authorized
14 for use in forestry in Ontario, having regard to the
15 nature of the assessments that you have described, are
16 safe from an ecological perspective if used properly in
17 accordance with authorized procedures?

18 A. I guess, along with Dr. Ritter, I
19 would have some concerns over just how we use safe. I
20 believe I would state it as, they do not cause
21 unacceptable environmental disturbance.

22 Q. And would you, like Dr. Ritter,
23 concur that, given the fact of their continuing
24 registration status, that members of the public are,
25 insofar as your discipline is concerned, entitled to

1 rely on that registration status as evidencing that
2 they are safe for use if used properly?

3 A. Yes, I would.

4 Q. Thank you very much.

5 MS. CRONK: Those are all my questions,
6 Mr. Chairman. Thank you.

7 THE CHAIRMAN: Thank you, Ms. Cronk.
8 Thank you, panel.

9 It is time to adjourn for the day. We
10 will adjourn until next week at 1:00 p.m.

11 Mr. Colborne?

12 MR. COLBORNE: Mr. Chairman, it has been
13 some time since I attended here and I notice that a
14 number of the faces have changed but, in any case, you
15 will recall that in October of last year I had the
16 pleasure of inviting everybody and hosting a social
17 gathering.

18 I know that almost everybody in this room
19 lives elsewhere and so I hosted what was billed as the
20 first semi-annual social gathering that had to do with
21 this hearing, and although I am not attending here
22 regularly, I have spoken to some of my fellow counsel
23 over the months and they have all encouraged me to have
24 the second semi-annual social gathering, and so I am
25 doing that.

1 I am doing it next Wednesday evening,
2 same place, and I believe that at least some of the
3 people who I have had a chance to speak to in the last
4 few days are available, including yourself, sir, and so
5 I am here to - on the record I suppose, or maybe it is
6 off the record - invite everybody and I hope everybody
7 can come.

8 Thank you.

9 THE CHAIRMAN: Thank you very much. We
10 will be pleased to attend.

11 I note that you refer to this as the
12 semi-annual one. We should perhaps be concerned at
13 this time next year if we are into our fourth or fifth
14 semi-annual one. But at this point we are delighted.

15 Thank you.

16 All right, ladies and gentlemen, we will
17 adjourn until 1:00 p.m. next Monday.

18 Thank you.

19 ---Whereupon the hearing adjourned at 12:20 p.m., to be
20 reconvened on Monday, August 14th, 1989, commencing
21 at 1:00 p.m.

